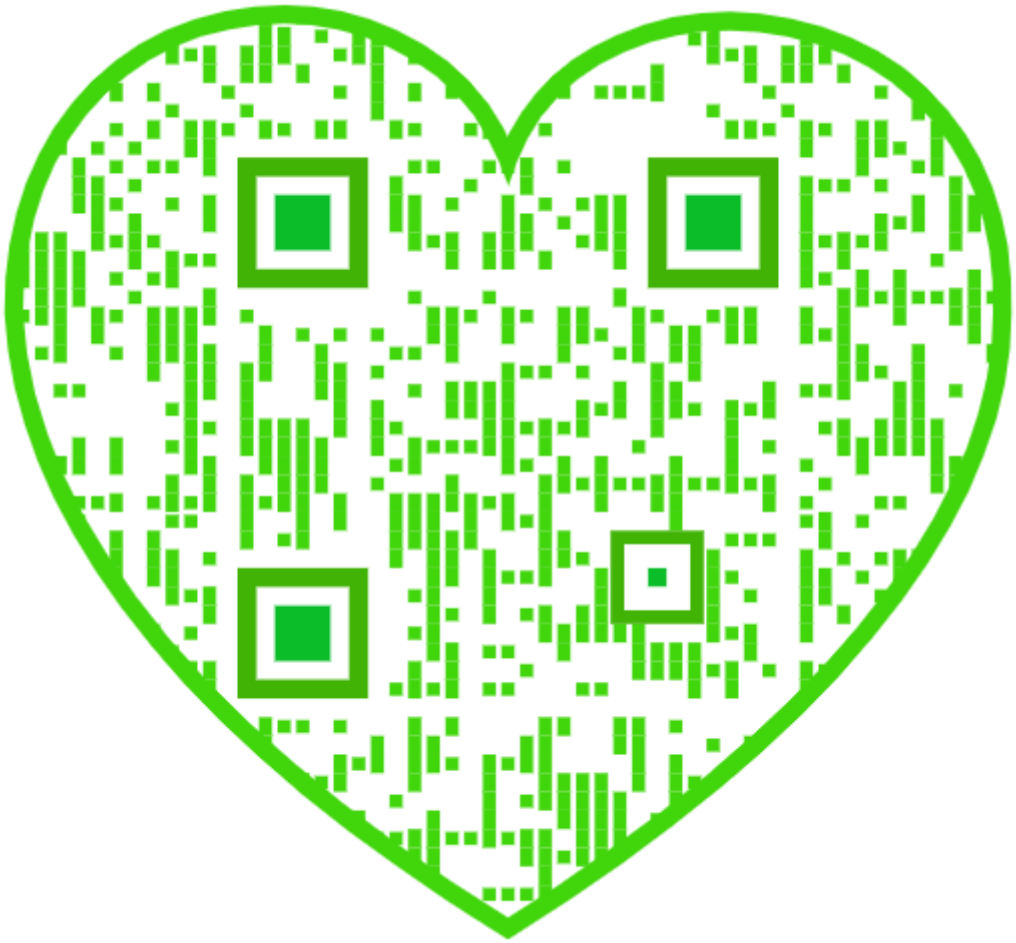

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Book .K5

THE

PRACTICE OF MEDICINE

A GUIDE TO THE NATURE, DISCRIMINATION AND MANAGEMENT OF DISEASE

BY

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TO MY WIFE

PREFACE.

The following pages represent an effort to prepare for the student and the junior practitioner of medicine a guide to the nature, discrimination, and management of disease that should contain the essentials unadorned with great detail. Since the determination of what is really essential must be a matter of individual judgment, the greatest problem encountered was what to exclude, so that the book, without exceeding a convenient limit, should be comprehensive, properly balanced, and in good perspective. An acquaintance with the present development and the trend of medical education, and a teacher's appreciation of the needs of students, make one realize the embarrassment of riches, the large number of facts, too often placed before minds not yet qualified to sort them in their order of importance and grasp their mutual bearings. This defect of medical pedagogy violates the physiology of the mind, which is so constituted that the natural stimulus to interest, and therefore to recollection, is the tracing of rational connection. The resulting mental indigestion and lethargy appear at examination time and when the student or junior practitioner is confronted with the necessity of applying at the bedside principles ill understood. A book, of course, cannot replace actual clinical experience, but it should in fairness offer its readers a well-ordered statement of existing knowledge as a basis for training their powers of observation and reasoning, especially in conformity with opportunities presented at the bedside.

These considerations determined me to devote most space and attention to the practical aspects of medicine; to the elucidation of those principles exemplified in disease at the bedside, in hospital wards and clinics, and in the consulting room; and to the clinically important and more common disorders rather than to the rarer diseases, however interesting. Such a plan seemed to offer more of value to students in recitations and in hospital-ward and dispensary work, and to practitioners desiring to recall elusive facts. A systematic arrangement of the subject matter has been adopted to facilitate comprehension of the mutual relations of different clinical and pathological entities. Emphasis has been laid upon the definition of processes of disease—to impress upon the student the necessity of accuracy in his use of terms and in his clinical descriptions. Inasmuch as the final object of medicine is practice, most space has been devoted to symptomatology, diagnosis, and treatment; and to ensure a rational grasp of these main objectives, they are presented in their natural relationship by means of brief

connecting sections on etiological factors, essential anatomical lesions, and pathological physiology. The sections on pathological physiology might have been considerably expanded; but I venture to hope that, although limited to a statement of the main facts, they may prove serviceable to students in affording them a broad view of disease and of disturbed functions in general. Throughout the book an endeavor has been made to portray the nature and natural history of disease, to correlate disturbed or perverted function with altered structure, to explain the development of symptoms, and to elucidate the sequence of cause and effect. Such knowledge is essential to a correct and intelligent comprehension and interpretation of morbid phenomena (symptomatology and diagnosis), to the ability to forecast and provide against contingencies, to judgment of the course, duration, and termination of disease (prognosis), and to skill in rational treatment and prophylaxis. Under the individual diseases, I have, as a rule, emphasized at least one method of treatment that I know to be of value from my own experience; beyond this point, however, I have depended upon the recommendations of acknowledged competent authorities. A few formulæ have been included as an aid to the junior practitioner; these with the other instructions mentioned should serve him in times of stress until his experience and judgment warrant his making his own modifications. Intentional limitation of space and the primarily clinical purpose of the book have made it seem desirable to omit discussions of the biological characteristics of the infectious microorganisms and of the technique of laboratory methods of diagnosis.

A book on such a boundless subject as Medicine must be evolved out of the collective experience of the masters of all times and all parts of the world, tinctured by the study, investigation, judgment, and experience of the author. Although this book, in large part, is based upon my own clinical and pathological experience, I must acknowledge my indebtedness to the many writers of many tongues whose contributions to medicine form the basis of my own knowledge. My experience with those disorders known as tropical has been almost limited to the rather few imported cases that an active hospital physician encounters in a large seaport. I have, nevertheless, included a brief account of these diseases, because of the widespread interest recently shown in their investigation, and on account of the increasing number of students who purpose practising in our tropical possessions; the clinical descriptions, however, are largely a reflection of those of such highly esteemed authorities as Manson, Cantlie, Jackson, and others. The descriptions of the metazoan parasites have been taken, in many instances verbatim, from Stiles—the recognized authority in this country. Elsewhere the attentive reader will note my indebtedness to other authors—to all of whom, I hope, I have given due credit.

A. O. J. K.

Philadelphia, 1910.

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THE PRACTICE OF MEDICINE.

SECTION I.

INFECTIOUS DISEASES.

The infectious diseases comprise a series of disorders that result from the growth within the living body of divers pathogenic vegetable and animal parasites. These, the so-called infectious agents (contagium vivum), include certain bacteria, certain non-bacterial fungi, and certain zoöparasites (protozoa, worms, and arthropods). In consequence, one may divide the known infectious diseases into three classes: (1) The bacterial infections; (2) the non-bacterial fungus infections; and (3) the zoöparasitic infections; but to these one must at present add a fourth class, that of infections of unknown or doubtful etiology—which comprise diseases undoubtedly infectious that have not yet yielded up to scientific inquiry their specific etiological factors. Perhaps the causative agents of many of these, as has been demonstrated of some of them (yellow fever, hydrophobia, epidemic poliomyelitis, pleuropneumonia of cattle, foot-and-mouth disease, etc.), are certain ultramicroscopic parasites, microörganisms that pass through a fine porous filter impervious to ordinary bacteria, but elude detection by the highest powers of the microscope. Additional reasons, however, for the non-discovery of these organisms are that they may be difficult to stain, difficult or impossible to cultivate in artificial media outside the body, and present in small numbers in the diseases they provoke.

Infectious and Contagious Diseases.—In times past the diseases now recognized as infectious were divided into two classes—the miasmatic and the contagious. The miasmatic diseases were those surmised to be due to some cause, a miasm, arising without the body and capable of provoking disease without the intervention of another individual thus affected; the contagious diseases were those caused by some factor, a contagion, developed within the diseased body and capable of transmitting the disease to another individual (man or animal). either directly or indirectly (through the medium of a third person or an

object, or the air). Since a miasm has become a myth of the past, the miasmatic diseases have completely disappeared; but the terms contagious and infectious still obtain. Perhaps a satisfactory distinction may be drawn between them: The term infectious diseases is the broader, and includes the contagious diseases. The term contagious was and still may be used to designate diseases readily transmissible from the sick to the well by direct or indirect contact; diseases which seem to be transmitted through the medium of the air and in which (as in smallpox, measles, scarlatina, etc.) a zone of "contagious atmosphere" seems to surround the patient; and diseases of which the causal infectious agent, or contagium vivum, seems to be ill-adapted to lead a prolonged existence outside the living body. This distinction, which is based upon the ability or lack of ability of the infectious agent to lead respectively a parasitic or a saprophytic existence, is largely artificial, since borderland cases occur and diseases ordinarily not contagious in the aforementioned sense may become so (pneumonia, typhoid fever, etc.). The distinction, however, has a practical bearing, in that the diseases thus called contagious may be effectually controlled by isolation of the patient and strict hygienic precautions, whereas those that may be called the non-contagious infectious diseases are not thus so readily controlled; but in this respect also borderland cases occur (some cases of typhoid fever, pertussis, measles, scarlatina, etc.). The terms transmissible and non-transmissible and communicable and non-communicable have been proposed to surmount the supposed difficulty —which also is largely artificial; the term infectious diseases is a satisfactory and all-inclusive term.

Transmission of the Infectious Diseases.—The records of antiquity, as well as of more modern times, definitely prove that many of the infectious diseases, notably plague, cholera, dysentery, typhoid fever, yellow fever, Malta fever, malaria, influenza, and many so-called tropical disorders, have a permanent habitat, are endemic, in certain parts of the world. Some of these diseases are seldom or never encountered beyond the confines of their permanent habitat; others, from time to time, under various influences, spread from these endemic centres to more or less distant regions, or they increase in number and perhaps virulence in regions where they prevail continuously—the disease becomes epidemic. The epidemic may be local, as in familial, house, school, barracks, jail, and town epidemics, or it may be widespread, extending throughout several adjoining States or countries; the disease even may become pandemic, as has frequently happened in connection with plague, cholera, influenza, etc., originating in the Orient.

The infection may be spread by aërial convection, by contamination of the water supply, by infected persons, by a third person, by fomites (caravans, cargoes, baggage, rags, money, especially along lines of travel and commerce, as well as public drinking vessels, personal clothing, bed-linen, room furnishings, table utensils, children's toys, etc.), and by domestic animals, rats, mosquitoes, flies, fleas, bedbugs, ticks,

and other insects. Each of these modes of infection may be active under varying circumstances; but from time immemorial special importance has been attributed to transmission by means of fomites. Unquestionably, fomites are of importance; but since it has been definitely proved that malaria and yellow fever are transmitted by the bites of mosquitoes, that the personal and bed-linen and even the discharges of yellow-fever patients are not infectious, and that plague is transmitted by means of fleas that infest plague-infected rats, the fomites theory has been robbed of much of its former importance; and since it also has been definitely proved that many persons convalescent from certain infectious diseases, as well as others long since recovered and apparently healthy, are unconscious "carriers" of the specific infectious agent (typhoid fever, dysentery, diphtheria, scarlatina, meningococcic infection, cholera, etc.), the prime importance of direct infection from person to person has become more fully realized. The simultaneous infection of many persons in a community suggests, as a rule, aërial convection of the infectious agent (as in influenza) or contamination of the water supply (as in typhoid fever, cholera, dysentery, etc.); but many, usually small, but also widespread, epidemics may often be traced to a single individual. In prophylaxis, to be presently mentioned, the significance of all modes of infection must be duly appreciated; but it is unwise to overestimate the significance of fomites, and to overlook the slight sore throat (diphtheria), persisting oral and nasal discharges (diphtheria and scarlatina), and the chronic carriers of typhoid and dysentery bacilli in the stools—all of which are frequently responsible for more or less serious and widespread, though usually local, epidemics.

Pathways of Infection.—In health, the blood, the viscera, and the tissues generally are free from pathogenic bacteria, although the bronchial lymph nodes may contain more or less quiescent tubercle bacilli and the liver partly disintegrated colon and perhaps other bacilli; but the skin, the nose, mouth, and throat, the intestinal tract, and the female genital tract harbor an extensive bacterial flora. This for the most part consists of non-pathogenic bacteria, largely organisms of putrefaction; but sometimes pathogenic bacteria also are found, leading a saprophytic existence—pyococcic on the skin and in the nose, mouth, and throat, Bacillus coli, Bacillus pyocyaneus, pyococci, various anaërobic bacteria, etc., in the intestinal tract, and so forth.

Infection of the body with pathogenic bacteria may occur during intra-uterine life—so-called congenital or transplacental infection; after birth infection may occur by way of the skin, especially through the hair follicles, the sebaceous or sweat glands, or abrasions or wounds; by way of the respiratory tract, the digestive tract, or the urogenital tract; or directly into the blood (direct inoculation, bites of insects, etc.). Bacteria that do not succumb in the body are eliminated largely through the intestinal and the urinary tracts (feces and urine), but some also are eliminated with discharges from the genital tract in women,

by expectoration, coughing, sneezing, etc., and from open, especially suppurating, wounds, etc. Outside the body the bacteria may die, chiefly under the influence of sunlight, desiccation (absence of suitable moisture), inappropriate temperature, and insufficient or inappropriate nourishment. Many bacteria, however, live for varying periods, especially if they pass into the spore stage, and contaminating the air, soil, or water, or attaching themselves to persons, places, or things, may subsequently give rise to new infections.

Nature and Lesions of the Infections.—When exposed to infection, the body may be in a receptive or a refractory state, that is, it may be susceptible to or immune to the infection. The severity of an ensuing infection is determined somewhat by the pathway of infection, but largely by the number and virulence or the infecting microörganisms, by the presence or absence of associated microörganisms (mixed, double, concurrent, or secondary infections), and by the resistance of the subject, that is, by a natural or acquired, active or passive, partial or complete immunity. This resistance or immunity, which is an expression of the phagocytic, antitoxic, and bactericidal properties of the blood, is of much importance, and serves to explain the exemption from infection of certain individuals exposed thereto. It may become reduced under varying circumstances, such as exposure to cold and dampness (catching cold), fatigue, improper or insufficient food, hemorrhage, and debilitating diseases like chronic cardiac and pulmonary disorders, diabetes, nephritis, malignant disease, etc. In the last-named conditions, secondary or terminal infections, due usually to the pyococci, Bacillus coli, etc., are common, and are often directly responsible for the fatal issue.

The pathogenic activity of the infectious bacteria is exerted almost exclusively through the production of specific poisons. These bacteria, therefore, are included with the substances known collectively as antigens (antibody generators), that is, organic agents, which, when introduced into the body, not only give rise to the presence in the blood serum of toxic substances, but also provoke the formation by the cells of the body of various antibodies which also pass over into the blood serum. These comprise: (1) Pathogenic bacteria; (2) the poisonous secretions or products of certain zoöparasites (Treponema pallidum, trypanosomes, etc.); (3) animal cells, extracts, or fluids (erythrocytes, blood serum, snake venom, cells or extracts of different organs, etc.); and (4) vegetable cells or extracts (ricin, abrin, etc.). The term toxin is often used to include all the poisonous products of bacteria, of which, however, several kinds may be distinguished, although they are not always separable the one from the other. Of the different toxins and antibodies, the following are the most important: (1) Toxins (extracellular toxins), products of the secretory (or excretory) activity of the bacteria (especially Bacillus diphtheriæ, Bacillus tetani, the pyococci, etc.). These are enzyme-like bodies, separable by filtration from the bacteria, and are rendered inactive by heating to 56° C.; they exert a destructive

(degenerative) action on the body cells; and they provoke the formation in the body of substances that inhibit their action, so-called antitoxins, with which they probably unite chemically. Some of these toxins have a special affinity for certain tissues—the tetanus toxin, the hydrophobia virus, and the lepra bacillus for the nervous system; the meningococcus for the meninges; the gonoccocus and its toxin for the serous lining of the joints, etc. (2) Endotoxins (intracellular toxins), either a part of or intimately bound to the bacteria, and liberated only upon lysis or destruction of the bacteria. These also exert a destructive (degenerative) action on the body cells; they probably give rise to little if any antitoxin formation, but they do provoke the formation in the body of bacteriolytic substances. (3) Mycoprotein (bacterial protein), the bacterial substance itself, the chief action of which seems to be exerted locally—in the production of inflammation and leukocytic infiltration (chemotaxis). (4) Lysins, substances that give rise to solution not only of the provoking bacteria, but also of the body cells—whence one speaks of bacteriolysins, cytolysins, especially hemolysins, endotheliolysins, hepatolysins, nephrolysins, etc. The lysins of certain bacteria have been definitely demonstrated, especially those of the staphylococci (staphylolysin), streptococci (streptolysin), Bacillus tetani (tetanolysin), Bacillus coli, etc. In infections with these bacteria an antilysin is formed, and may be found in the blood serum. For the occurrence of an effective lytic action, such as hemolysis, it is necessary to have the interaction of an antigen (bacteria, alien erythrocytes, etc.), specific antibodies (so-called immune bodies, or amboceptors), and an active complement (completing substance, cytase, alexin) which is present in the blood serum of all normal animals. The union of the amboceptor and the complement constitute the lysin. (5) Agglutinins, substances that give rise to the agglutination or clumping of the specific infecting microörganism, as well as the agglutination or fusion of erythrocytes. The agglutination of pathogenic microorganisms constitutes the so-called Pfeiffer phenomenon, and is utilized in the Gruber-Widal reaction, of much diagnostic importance in typhoid fever, paratyphoid fever, bacillary dysentery, Malta fever, etc. Agglutination or fusion of erythrocytes is believed to be the cause of certain hyaline thrombi found in typhoid fever and other disorders. (6) Precipitins, substances that lead to the precipitation of bacteria, or to the formation of a sediment (precipitate) when added to a solution of the antigen that provoked their formation. This forms the basis of a test serviceable in the recognition of human blood. If a foreign protein, such as human blood serum, be injected into a rabbit, the rabbit's blood serum is presently found to contain a substance (precipitin) which, when the rabbit's serum is added to a suspension of some human blood (the one-thousandth part of a milligram or less), gives rise to a visible precipitate; this phenomenon does not occur if the rabbit's serum is added to other than human serum, except that of some of the anthropoid apes, and with these the reaction does not occur in such

high dilutions as with human serum. (7) Opsonins, substances that so act upon the infecting bacteria that they are readily destroyed by the phagocytes. (8) Aggressins, substances that act upon the phagocytes and other protecting agencies so as to inhibit or destroy their power of protecting the body from infection. In addition to the aforementioned predominant toxic influences, bacteria may provoke, usually relatively minor lesions, directly by obstructing small capillary or other vascular areas (bacterial emboli), or by being present in excessive numbers in the circulating blood (anthrax).

An established infection may be localized, that is, confined to the point of inoculation or some other point whence, immediately following inoculation, the bacteria are transported, or it may become generalized. In some local infections the body generally may be not at all affected, although the local lesions may increase somewhat in extent by continuity or contiguity of tissue. In other cases, the toxins formed locally gain access to the blood stream and the lymph stream and occasion a toxemia (bacterial intoxication) and certain general lesions and symptoms; this is especially marked in typhoid fever, diphtheria, scarlatina, tetanus, pyococcic infections, variola, etc. In still other cases the bacteria themselves invade the blood stream or the lymph stream, and give rise to a general hematogenous or lymphogenous infection (as the case may be), to a so-called bacteremia; this is notably the case in typhoid fever, syphilis, glanders, anthrax, many pyococcic infections, etc. Although some infections, such as tetanus, diphtheria, tuberculosis, etc., are almost if not quite always pure toxemias, and others, such as typhoid fever, syphilis, anthrax, etc., are always bacteremias, in the majority of infections no sharp line of demarcation can be drawn between toxemias and bacteremias. In most cases, as already stated, the lesions and symptoms are due to the bacterial intoxication, but bacteremia is much more common than is generally supposed; the bacteria, being carried to the different organs of the body, set up disseminated foci of disease (so-called bacterial metastases), analogous to those of the primary lesion.

The local and general results of infection vary with the nature of the infecting microörganism and its toxin, and with the tissues especially involved. In general, the local results consist of toxic degeneration of the cells and tissues, which may progress to complete necrosis, and of reactive changes comprised within the term inflammation. The inflammatory phenomena may be simple and non-specific, and serous, serofibrinous, fibrinous, purulent, or hemorrhagic in character, the same bacterium under varying circumstances giving rise to varying grades of exudation or proliferation; in other cases, the inflammatory phenomena may be specific, that is, so characteristic as to enable one thereby to postulate the nature of the initiating microörganism (tuberculosis, syphilis, glanders, etc.). In chronic infections there is likely to be also considerable formation of new connective or cicatricial tissue.

The general results of bacterial intoxication vary considerably in

different cases, but the most constant and conspicuous lesions consist of parenchymatous and sometimes fatty degeneration of the viscera, especially the myocardium, the liver, the kidneys, and the muscles. In addition, one may encounter focal necroses of the liver and the lymphoid tissues (the lymph nodes, the spleen, and the bone marrow), the result of the local action of the toxins or of occlusion of small vascular areas by large phagocytic cells or thrombi made up of fused erythrocytes (the consequence of the action of hemagglutinins). The spleen often becomes considerably enlarged, due in part at least to hyperplasia and multiplication of the endothelial cells of the splenic pulp—which cells become phagocytic and are perhaps otherwise concerned in the production of immunizing bodies (as in typhoid fever, for instance); in part the enlargement of the spleen is due also to the accumulation of large numbers of degenerated erythrocytes (the consequence of the action of hemolysins). In some cases degenerative changes occur in the nerves. In other cases small hemorrhages develop beneath the skin or mucous membranes or in the tissues, especially in typhus fever, variola, cerebrospinal fever, scarlatina, pyococcic infections, etc.; in some of these cases, notably the pyococcic infections (so-called malignant endocarditis, etc.), the hemorrhage often results from rupture of miliary (so-called mycotic) aneurysms, whereas in other cases the hemorrhage occurs in consequence of the action of an endotheliolysin that destroys the cells of the capillaries and the lining of the small arterioles, and thus permits the extravasation of blood. In many infections, changes in the composition of the blood are conspicuous, notable anemia and leukocytosis; these may be associated with changes (usually delay) in the clotting time of the blood and various ill-understood alterations in the constitution of the plasma. The anemia is due at first to increased hemolysis, but later often to interference with the erythroblastic function of the bone marrow (defective hemogenesis). The leukocytosis is an active process, commonly attributed to chemotaxis, and the result largely of proliferation of the leukoblastic tissues of the bone marrow; it is a beneficial process, constituting, together with the phagocytic, antitoxic, and bactericidal properties of the blood, the process whereby the body becomes protected against or immune to infections. Leukocytosis is notably absent in typhoid fever, malaria, measles, rötheln, influenza, mumps, the early stages of tuberculosis (except the meningeal form), and leprosy. The cause of this is not well understood, but it is attributable, in typhoid fever at least, to degenerative changes in the bone marrow; in other cases it may be due to the absence from the blood serum of a positively chemotactic substance.

Pathological Physiology of the Infections.—The evidence of reaction on the part of the body to an established infection, that is, the onset of symptoms, may occur soon after the infection has taken place, or it may be delayed for a variable, even long, time. It is delayed until the toxins are present in such amount and virulence as to overcome

the neutralizing or antagonistic forces of the body, to disturb the functional activity, and compromise the structural integrity, of the constituent cells. This period, that is, the period that intervenes between the time the pathogenic microörganisms begin to increase and multiply within the living body and the onset of symptoms, is known as the period of incubation. This varies considerably in duration in different diseases and somewhat in different cases of the same disease; it may be apparently twelve hours or less in some cases of pneumococcic infection of the lung (croupous pneumonia), and as much as two to six or more months in hydrophobia. In many infections this period is unannounced by symptoms; but in some infections, notably typhoid fever, toward the end of this period, concurrently with the diffusion throughout the body of some toxins or the dissemination of the causative bacteria, certain more or less ill-defined, so-called prodromal or premonitory, symptoms become manifest, such as malaise, lassitude, muscular weakness, and readily induced weariness, general neuromuscular pains and soreness, headache, mental hebetude or irritability, perhaps even slight rise of bodily temperature, etc.

The onset of definite symptoms, the so-called period of invasion, may be abrupt (acute) or insidious (subacute or chronic). In certain acute infections, such as croupous pneumonia, malaria, etc., the period of onset may usually be determined with great exactitude, the invasion symptoms consisting of a more or less severe chill and fever. Similar, but less aggravated phenomena may attend the onset of other acute infections; in infants and children the initial chill or chilliness is often replaced by a convulsion. In infections of more insidious development, such may be the gradual increment of symptoms that it may be almost if not quite impossible to determine accurately the time of onset of the disease. In infections generally, of all the symptoms of invasion, fever is the most constant and most significant. Such, in point of fact, is its significance that in the great majority of cases its occurrence should be interpreted to mean infection or intoxication; other less common causes comprise the action of enzymes such as are liberated during hemolysis (internal hemorrhages, certain severe anemic states, burns, etc.), the excessive action of internal secretions (thyroid), grave perturbations of the central nervous system associated with increased heat production or lessened heat dissipation, exposure to high external temperature, and certain drugs (strychnine poisoning, etc.).

Following upon the period of invasion, the infections are characterized by a more or less prolonged period of fever, the so-called fastigium—which may last several hours, several days, or several months or more. The type and course of the infection may be acute, subacute, or chronic. The acute infections, such as measles, scarlatina, smallpox, croupous pneumonia, typhoid fever, relapsing fever, etc., are characterized by a definite period of incubation, a characteristic, often abrupt period of onset, a definite fastigium (typical fever course), and often quite characteristic if not pathognomonic symptoms. Depending upon the

virulence of the infectious agent and the resistance of the subject, recovery or death eventually occurs. In favorable cases, following the fastigium, defervescence ensues—the temperature falls either suddenly (crisis) or slowly (lysis), the other symptoms abate, and convalescence becomes established. The convalescence, however, may be interrupted by relapses or complications (often secondary infections), and restoration to complete health may be delayed by sequels. Certain of the acute infections are sometimes characterized by dominant intoxication, marked mental depression or coma, slight fever or even subnormal temperature (that is, little or no reaction), rapid and feeble pulse, low and falling blood pressure, absence of leukocytosis in diseases in which it ordinarily occurs, and early death. These cases are often spoken of as hyperacute, fulminating, or malignant; they are sometimes exemplified in smallpox, cholera, yellow fever, plague, pyococcic (especially streptococcic) infections, cerebrospinal fever, scarlatina, malaria, etc. The chronic infections, such as tuberculosis, glanders, actinomycosis, etc., as a rule, are characterized by a variable period of incubation, although syphilis is a noteworthy exception, having a definite period of incubation; as a rule, also, they are insidious in onset, a distinct period of invasion often being undeterminable; and their course is protracted. To intermediate cases the term subacute is often applied; other cases, characterized by periods of remission and of relapse or exacerbation, are spoken of as remittent or recurring. Finally, Adami has postulated what he describes as subinfection, a condition in which pathogenic and non-pathogenic bacteria, instead of proliferating, are destroyed in the various tissues and organs, especially the liver and the mesenteric lymph nodes, to which they may be carried by the lymph and blood stream. The long continuation of this process is believed by Adami to lead ultimately to cell exhaustion and to be perhaps a factor in the production of cirrhosis of the liver, some cases of pernicious anemia, some cases of chronic interstitial nephritis—an opinion that has not yet found confirmation.

The obtrusive characteristic of the infections is fever; but increased temperature is only one feature of the febrile state. Although under the influence of bacterial intoxication the thermogenic regulating centres are much disturbed, the increased temperature is due largely, if not wholly, to increased heat production, which in turn results from augmented tissue katabolism and oxidation; that is, under the influence of bacterial toxins, and independently of the amount and character of the food, tissue destruction is greater than tissue formation; the tissue destruction, however, may be somewhat controlled by appropriate food. Adami looks upon fever as the process of adaptation to such agencies as can be neutralized by the development of antibodies. Since it represents the reaction of the body to the infection, and in an otherwise toxic case is to some extent a measure of that reaction, it is by no means an unmixed evil; it shows that the body is coping better with the infection than if fever did not occur. Although hyperthermia,

especially if prolonged, is of itself dangerous to life, the toxic apyretic cases, particularly cases in which the manifestations of intoxication increase while the temperature falls, are really serious and commonly terminate fatally.

As already stated, the fever of invasion is often accompanied by a more or less severe chill; this is the more likely to occur if the initial (pyrogenetic) stage of the fever is short—from one-half to two hours; in other cases there may be only chilliness, which may last for some time or be repeated. The chill is a nervous manifestation, characterized by subjective sensations of coldness, chattering of the teeth, fibrillary twitchings of the muscles, and pallor and lividity of the face and extremities. Although the patient feels cold and complains thereof, the internal temperature of the body is increased. The condition is attributable to contraction of the peripheral bloodvessels under the influence of vasomotor stimulation, in consequence of which the surface of the body is ill-supplied with blood, while the internal organs are congested; the fibrillary twitchings and the increased tone (rigor) of the muscles are doubtless in part responsible for the augmented heat production.

In addition, the febrile state is characterized by other nervous manifestations, as well as disturbances of the circulatory, respiratory, digestive, and urinary systems, and disorders of general metabolism. The nervous manifestations may be irritative or depressive: the irritative, which usually precede the depressive, consist of general neuromuscular pains and soreness, headache, mental irritability, intolerance of light and noises, insomnia, active delirium, etc.; the depressive consist of mental hebetude, apathy, inattention to surroundings, retention of urine or involuntary evacuation of the bladder and rectum, muttering delirium, stupor, coma, etc. These are attributable to a direct action of toxins on the central nervous system, which at first is irritated and finally exhausted. In some cases nervous symptoms are unusually dominant and give rise to so-called cerebral types of several infections; these very much simulate meningitis, and constitute the so-called meningism of the French. In other cases, such as tetanus, hydrophobia, epidemic poliomyelitis, etc., the toxin (or virus) possesses a special affinity for the nervous system, and may reach the central nervous organs by way of the peripheral nerves.

Rapidity of the cardiac action and of the pulse is an almost constant concomitant of the febrile state; as a rule, each degree of added temperature corresponds to about ten added cardiac beats per minute, but there are exceptions: in typhoid fever, for instance, the pulse is commonly disproportionately infrequent, in scarlatina it is characteristically disproportionately rapid, and in the event of stimulation of the pneumogastric nerve (as in meningitis), it may be very infrequent. In addition to being rapid, the pulse may be full and bounding, with normal or perhaps increased blood pressure (in sthenic states); or weak, easily compressible, or thready, with lowered blood pressure (in asthenic

states). Increasing rate and weakness are of bad prognostic omen, whereas decreasing rate and increasing strength render the outlook hopeful. These changes are the result of various factors: perhaps early there is toxic stimulation of the accelerator nerves and increased excitability of the heart, and consequent increased pulse rate; later toxic degeneration of the myocardium occurs and leads to depression of contractility and tonicity and more or less ineffectual systoles; toxic degeneration of the pneumogastric nerves may be answerable for some of the sudden deaths that occur during the course of or in convalescence from diphtheria, influenza, etc.; and toxic paralysis of the vasomotors is responsible for the lowered blood pressure and the common capillary stasis, especially of the fingers, toes, nose, ears, etc.

Furthermore, the respiratory rate and the respiratory exchange of gases become increased. The increased respirations are due to a direct action of the toxins and the augmented temperature on the respiratory centres and to surcharging of the blood with carbon dioxide; the increased gaseous exchange in the lungs is a direct consequence of the increased metabolism, of the increased formation of carbon dioxide, and the necessity for more oxygen. The increased respirations serve in a measure to reduce the bodily temperature, some of which is dissipated in the process of warming the increased amount of air inhaled. The chief digestive disturbances consist of anorexia, dry mouth, thirst, lessening of the digestive juices, and often constipation; the gastric juice, as a rule, is lessened in amount and reduced in acidity; the bile becomes inspissated and is perhaps otherwise altered; the intestinal juices are reduced in amount, which together with diminished peristalsis is answerable for the constipation of some cases. The urine becomes reduced in amount, concentrated, of heightened color, increased acidity, increased specific gravity, and increased toxicity; it contains an increased amount of nitrogenous substances (urea, uric acid, kreatinine, etc.) and of potassium, magnesium, and calcium salts, but sometimes there is a decrease of the chlorides; there is often also a small amount of albumin, and sometimes hyaline and other casts; in the event of intestinal putrefaction, decomposition products, such as indol, skatol, and other aromatic substances, may be encountered. The significant change from the normal is the great increase in the nitrogenous excretion, which may reach twice or thrice that of a normal person on a like diet; this is the result of the augmented katabolism affecting especially the proteins of the body. One evidence of this is seen in the emaciation of the infected subject—at first the panniculus adiposus disappears, but the muscles soon decrease in size, lose their tone, and become soft, flabby, and readily exhausted.

The foregoing statements in reference to the bacterial infections apply with equal force, although in somewhat modified form, to the non-bacterial fungus infections and the zoöparasitic infections. Some of the protozoan infections, such as malaria, syphilis, relapsing fever, trypanosomiasis, etc., exhibit many analogies with bacterial infections—

the fever, toxemia, hemolysis, disturbances of metabolism, etc. Some of the metazoan infections also seem to be associated with the production of a hemolytic and perhaps other poisons, and the metazoa often serve also as hosts carrying pathogenic bacteria into the human subject. Many questions concerning the zoöparasitic infections, however, await elucidation, not only those pertaining to etiological factors and the modes of transmission, but also those relating to natural and acquired immunity.

Diagnosis.—Many of the infections may be recognized with certainty, others with relative certainty, by means of their clinical manifestations—which are discussed under the respective headings. In addition, the progress of medical science during recent years has rendered available several more or less specific diagnostic methods. Many infections of known etiology may be diagnosticated without doubt by isolating from the patient the specific causative microörganism. This may be found on or in more or less superficial lesions of the skin or accessible mucous membranes, in the blood, in the cerebrospinal fluid, in fluid removed by puncture from the serous sacs, in the organs and tissues of the body, in discharges, etc.; and the microörganisms may be studied in coverslip preparations or by cultural methods. As already stated, the agglutinins are the active factor in causing agglutination of bacteria, a phenomenon utilized in the Gruber-Widal reaction—the agglutination or clumping of microörganisms when added, in dilutions of 1 to 50 to 1 to 1000 or more, to blood serum containing specific agglutinins. This is an important diagnostic test in typhoid fever, paratyphoid fever, relapsing fever, Malta fever, bacillary dysentery, cholera, plague, glanders, tuberculosis, etc. The presence of bound or unbound complement is utilized as a basis of the Wassermann complement-binding (or deviation-of-the-complement) test in the diagnosis of syphilis and so-called parasyphilitic disorders, like tabes dorsalis and paretic dementia; its use has been suggested also in typhoid fever, meningococcic and gonococcic infections, trypanosomiasis, echinococcic disease, carcinoma, etc. The reaction, which is based upon original observations of Bordet and Gengou, requires for its successful carrying out much experience and laboratory facilities. In brief, it consists of adding to the blood serum of a patient suspected of being syphilitic a watery extract of a syphilitic (usually fœtal) liver (or other organ containing Treponema pallidum); if the patient is syphilitic, the blood serum contains syphilitic antibodies or amboceptors and the complement is bound. If this mixture be now added to sheep's erythrocytes and the corresponding amboceptors, hemolysis does not occur—the available complement (necessary to complete the hemolysis) being already bound. Recently it has been found that extracts of normal liver, sodium glycocholate, lecithin, and other lipoids may be utilized instead of the original antigen (syphilitic liver); and more recently Noguchi has modified the test by substituting an antihuman for the original antisheep hemolytic system, and by utilizing test papers

saturated with antigen, amboceptor, and complement. Finally, it has been found that under the influence of bacterial toxins and other antigens, certain animals and human subjects develop a specific hypersensitiveness to the antigen in question (anaphylaxis), so that a second injection of the antigen may bring about serious and sometimes fatal results, although the two doses combined were less than that ordinarily provocative of toxic results. Such a phenomenon is sometimes observed in human subjects after the injection of diphtheria and other antitoxins, the serum and not the antitoxin as such being the active factor (serum disease or fatality). In tuberculosis, glanders, and other infections the tissues also become sensitized to the toxin in question, so that when an extra amount is added they react variously; this is the basis of the various tuberculin reactions, the mallein reaction, etc.

Prophylaxis.—The prevention of the infections is no less important than their cure, and since each case presupposes an antecedent case, efforts should be made to throttle the disease at its source. This, the individual patient, is the practising physician's chief concern. The patient should be isolated. So imperative is this in infections, such as smallpox, scarlatina, diphtheria, typhus fever, yellow fever, cholera, plague, etc., that isolation and supervision is one of the readily conceded functions of the health authorities, is ordered by law, and in the best regulated communities is effected in special hospitals provided for the purpose. It is equally desirable that isolation should be practised in other infections, which if they do not create the communal consternation that pertain to those already mentioned, still have a greater morbidity and often a larger death toll—that is, typhoid fever, measles, rubella, pertussis, cerebrospinal fever, puerperal fever, pneumonia, tuberculosis, gonococcic infection, syphilis, etc. If treated at his home, the patient should be placed in a large, clean, quiet room, provided with an abundance of sunlight and fresh air, and adequately protected against flies, mosquitoes, and other insects; all unnecessary furniture, carpets, hangings, pictures, etc., should be removed; and a sheet, wet with carbolic acid, mercuric bichloride, or formalin, may be hung before the door, if for no better purpose than to warn away the inquisitive. In the readily transmissible infections (diphtheria, scarlatina, smallpox, etc.) the patient should be provided with at least one, preferably two, nurses or attendants; all other persons should be rigidly excluded from the sick room. The nurses, as well as the physician, prior to entering the sick room, should don a gown that entirely envelops the body and should cover the head; and on leaving the room and removing the gown, they should wash the exposed parts of the body (hands and face). The nurses should also wash their hands after touching or handling the patient; or they may protect themselves by wearing rubber gloves. Similar precautions should be adopted also in the less readily transmitted infections, notably typhoid fever, tuberculosis, etc.

In all cases of infection, the patient's personal linen, bed-linen, dishes, knives, forks, spoons, etc., should under no circumstances

be placed in contact with those used by other members of the household; nor should the attendants upon the patient prepare food for other members of the family. All dishes and other utensils used by the patient (such as the bed-pan, urinal, etc.), should be disinfected after use, by boiling or the use of a 5 per cent. carbolic acid solution. All discharges from the patient, such as from the mouth, nose, or ear, the urine, feces, sputum, vomitus, etc., should be destroyed: if possible, by incineration and complete destruction, or, when this is not available, by the use of milk of lime, carbolic acid (5 per cent.), or mercuric bichloride (1 to 500 or 1 to 1000). The milk of lime must be freshly prepared by adding to a lump of quicklime about half its volume of water; the resulting hydrate should be kept sealed, and as required one part of it should be added to four to six parts of water. Whichever disinfecting solution is used should be added to the discharge in the proportion of two to three volumes to one, thoroughly mixed, and allowed to act for two to six hours before being emptied into the closet. Throughout the course of the infection, the patient's personal and bed-linen, etc., should be put directly from the bed into a large sheet and then into carbolic acid (5 per cent.), or mercuric bichloride (1 to 500); thence it should be removed to a suitable place and boiled for at least one hour.

After the patient has recovered, he may be given a bichloride bath (1 to 2000), and his skin may be anointed with olive oil; he should then be removed to another room, previously well aired, and fresh linen should be provided. Should death occur, the body should be enveloped in several sheets or other cloths wet with carbolic acid (5 per cent.) or mercuric bichloride (1 to 500), and as soon as possible placed in a coffin and hermetically sealed. After convalescence (or death) the room and its contents must be thoroughly disinfected. The preferable method for the contents is by incineration and complete destruction; this is desirable especially for old mattresses, carpets, rugs, hangings, and other articles of little value; articles of value may be subjected to the action of superheated steam (steam under pressure), or boiling water, or chemicals, such as carbolic acid, mercuric bichloride, or formaldehyde. The room itself should be hermetically sealed and subjected to the action of formaldehyde gas, which may be generated in special generators, or by the addition of potassium permanganate to the official formalin, in the proportion of 8 ounces (250 grams) of the permanganate to one pint (500 c.c.) of the formalin to each 1600 cubic feet of air space. The room should not be opened until after the lapse of twelve to twenty-four hours. Thereupon the floor and other woodwork, etc., should be scrubbed with hot water and soap, and then washed with mercuric bichloride (1 to 500)—which some time after it has dried should itself be washed off. The room then should be exposed for a day or two to a vigorous current of fresh air and as much sunlight as possible. New wall paper should be substituted for the old.

In addition to the aforementioned measures, which are designed to prevent the spread of infection, there are other important prophylactic procedures. These comprise, particularly, a pure, non-infected water supply, which is especially important as regards typhoid fever, dysentery, cholera, etc. The water should be taken from a source as pure as possible, and in the event of even the slightest doubt, should be filtered (chemical disinfection does not seem to be as efficacious as filtration, and is otherwise objectionable). During the prevalence of an epidemic all the drinking water should be boiled, and only boiled water should be used for washing the teeth, as well as foodstuffs, like celery, lettuce, etc. Special care must be taken to see that other articles of diet are not contaminated; such as milk (diluted with infected water or handled in dairies where an infection prevails); oysters and other shellfish grown in polluted streams; vegetables grown in infected ditches or watered or washed with infected water; meats from diseased animals, etc. These if eaten at all must be thoroughly cooked. Attention must be paid to the existence of chronic "carriers," who are often responsible for local or widespread epidemics of typhoid fever, dysentery, etc., as well to the slight sore throat (diphtheria, scarlatina) and the persisting nasal and aural discharges that often follow diphtheria, scarlatina, etc. In time of war or during peaceful military encampments, additional sanitary measures should be undertaken, especially the proper disposal of refuse, urine, and feces, the protection of latrines and garbage cans from flies and other insects, etc. An efficient prophylaxis of the large and increasing number of ill-understood infections in the tropical and subtropical regions, some of demonstrated protozoan origin and many known to be transmitted by various insects, is to be expected from a careful study of the life history of the infection-carrying insects, and the adopting of preventive measures in accordance with the discoverable facts. Finally, among the prophylactic measures, one must mention preventive inoculations, which are resorted to with complete success in smallpox (vaccinia), and have been used with encouraging results in hydrophobia, typhoid fever, cholera, plague, anthrax in the lower animals, etc.

Treatment.—Since the specific nature of many infections has been demonstrated, scientists have labored to produce specific remedies that shall, in some measure, at least, reproduce the normal immunizing—antitoxic, bacteriolytic, and phagocytic—processes of the body. Some are available and of unquestionable utility. Diphtheria antitoxin, the first discovered, has virtually revolutionized the treatment of diphtheria, and may be looked upon as a specific, certain to cure if resorted to early in the course of the infection. The hope born of this success has not found fulfilment as regards many of the other infections—because antitoxin is efficacious only if resorted to before the toxin has formed a firm union with or destroyed the tissues, and because many of the bacteria produce not an extracellular toxin, but an intracellular, so-called endotoxin, which does not provoke the formation of an anti-

toxin, although it may lead to the formation of a bacteriolytic substance. The tetanus antitoxin, for instance, is usually inefficacious, because the development of symptoms is significant of a firm, almost indissoluble union between the toxin and the nervous tissues. Success has attended the prophylactic use of this tetanus antitoxin in cases, such as Fourth-of-July wounds, known to be especially likely to be followed by tetanus. The use of pneumococcic, staphylococcic, streptococcic, and plague antitoxin, as a rule, has not been followed by results better than those which attend other forms of treatment, although some notable successes have been achieved. Encouraging results have followed the use of Flexner's antimeningococcic serum. The use of bacteriolytic serums is not to be unreservedly commended, since the solution of bacteria in the tissues is not free from danger. Recently bacterins (bacterial vaccines; suspensions in physiological saline solution of killed bacteria) have been used with considerable success, especially in staphylococcic, streptococcic, pneumococcic, gonococcic, tuberculous, dysenteric, some typhoid-colon infections, Malta fever, etc. These bacterins act by facilitating or augmenting an active immunity, and while they doubtless favor the production of several immune substances, their chief action perhaps is to stimulate the production of specific opsonins, and thus to facilitate phagocytosis. Wright believes that the administration of bacterins should be controlled by estimations of the opsonic index (the opsonic power of a patient's blood, as compared with that of a healthy person, in respect to a given microörganism); this in practice, however, has not been found necessary. Autogenous vaccines are much more efficacious than stock vaccines. Recently also Hiss, acting upon the hypothesis that the activity of the phagocytes in the processes of immunity is due, in part at least, to the presence of an intracellular antitoxin (endo-antitoxin), has suggested the use of aqueous extracts of washed dead leukocytes in staphylococcic, streptococcic, pneumococcic, meningococcic, and other infections. Encouraging reports of the efficacy of the treatment have been made. But all methods of specific therapy are still on trial; the final pronouncement regarding their value is reserved for the future.

BACTERIAL INFECTIONS.

TYPHOID FEVER.

(*Enteric Fever; Abdominal Typhus.*)

Typhoid fever is an acute, specific, infectious disease caused by Bacillus typhosus (Eberth-Gaffky); it is a true bacteremia, characterized anatomically by hyperplasia and necrosis of the lymphoid tissues of the body, especially of the intestinal tract, and clinically, by slow onset, continued fever of characteristic type, enlargement of the spleen, a

characteristic roseolous rash, a specific serum reaction, the so-called typhoid state, and a tendency to relapse and to the occurrence of certain complications, notably intestinal hemorrhage and perforation.

Etiology.—While typhoid fever occurs at all ages, even in infancy and in old age, it is especially frequent between the ages of fifteen and thirty years; but typhoid infection may occur even *in utero*. It is equally common in both sexes, and affects persons in all stations of life. It occurs in all parts of the world, but is especially prevalent in the temperate and subtropical zones. It is endemic in most large cities and frequently prevails epidemically, especially in the autumn and early winter—more commonly after a dry, hot summer (whence the term autumnal fever); but in some localities the disease is quite frequent in the spring, concurrent with the thaws and floods. Newcomers in infected localities are especially susceptible, more particularly those who move from the country to the city. Bad sanitary arrangements, familial and municipal, such as defective drainage, contaminated water supply, poor ventilation, the accumulation of filth, etc., act as predisposing factors—favoring the development of the typhoid bacillus and reducing the vitality of the individual. Pettenkofer attributed the development of the disease to the condition of the "ground soil," believing that a low level of the standing water favors the occurrence of the disease. One attack usually confers more or less absolute immunity, but recurrences are by no means unknown. Some persons seem to possess a natural immunity. The immunity of old residents in infected localities, however, is frequently due to their having had the disease in early life—when the symptoms, as a rule, are mild and the mortality low.

The exciting cause of the disease is Bacillus typhosus. Infection is almost exclusively transmitted by infected food or infected drink; sometimes by direct contact, by a third person, by the air, by fomites, or by infected animals. Every case of the disease thus presupposes the existence of some previous case, that directly or indirectly acts as a source of infection; many widespread and fatal epidemics have been traced to a single case. Infection is commonly acquired by way of the gastro-intestinal tract; rarely perhaps by way of the respiratory tract. In the usual, that is, the gastro-intestinal cases, the infectious atrium has not been definitely determined, the ileum, the upper part of the intestine, the stomach, and the tonsils being variously looked upon as the portal of entry of the bacillus. For obvious and good reasons the ileum is generally believed to be the portal of entry, but as opposed to the correctness of this view one may perhaps adduce the early bacteremia (as early as the fourth day) when changes in the lymphoid follicles of the ileum are slight, and the early disappearance of bacilli from the feces: they are believed not to thrive well in the intestinal contents and those present not improbably come from the gall-bladder and the bile into which they are promptly excreted from the blood. Infection by way of the gastro-intestinal tract results from the swallowing of infected food or drink, especially water, milk, oysters, and the

like. The drinking water usually becomes contaminated by soakage of infected sewage, feces, or urine into wells, cisterns, springs, reservoirs, rivers, etc. Milk is usually contaminated by being diluted with infected water, or by being put into utensils washed with infected water; or it becomes infected from the unclean hands of dairy attendants or other persons. Derivatives of milk, such as cream, ice cream, butter, etc., may also serve as sources of infection, as well as articles of food that have been washed with contaminated water, or that may be eaten raw, such as oysters fattened in polluted streams, and lettuce, celery, etc., grown in contaminated ditches or watered with contaminated water. Food may also become infected by flies and other insects, to whose feet the bacillus readily adheres or whose feces may contain the specific germ. This occurs especially in rural districts, the family privy vault often being the source of infection; in warm climates; and during army movements, the flies conveying the bacillus directly from latrines to the kitchen or mess table.

Direct transmission from the sick to the well sometimes occurs (one-fourth of the cases Conradi), and is apparently the only cause that can be assigned for some cases of the disease occurring in nurses, attendants, etc. The emanations from the patient are not necessarily infective; but the stools, urine, etc., are not properly disinfected, and infect the bed-clothing, the patient's personal linen, etc., and these being incautiously handled, serve as the indirect source of infection. A number of cases, even restricted epidemics, have recently been traced to so-called "carriers," subjects of past typhoid fever, whose feces may contain the typhoid bacillus continuously or intermittently (chronic or intermittent carriers). The feces become contaminated from the gall-bladder, which may remain a permanent habitat of the bacillus.

Infection by way of the respiratory tract is relatively uncommon. That it is possible cannot be denied (some cases of apparently primary typhoid pneumonitis). However, bacilli in the air being inhaled are usually arrested in the pharynx and eventually gain the gastro-intestinal tract—whence infection occurs, as is usual. It is possible that in rare instances infection may occur through the tonsil, and the blood thus become primarily infected (certain cases of typhoid fever without intestinal lesions?).

Pathology.—The characteristic macroscopic lesions of typhoid fever involve the solitary and agminate lymph follicles of the intestine, the mesenteric and other lymph nodes, and the spleen. The typhoid bacilli having reached the intestine doubtless invade the lymphoid tissues, the solitary and agminate follicles, thence the mesenteric lymph nodes, and ultimately become dispersed through the lymphatic and the blood circulation and all the organs of the body. The changes in the lymph follicles of the intestine are commonly said to exhibit four stages—infiltration, necrosis, ulceration, and cicatrization—which correspond approximately to the four weeks of the disease, but approximately only, since the different processes may be shortened or markedly prolonged, and in some cases

the lesions may not proceed beyond infiltration, resolution occurring soon. At the end of the first week, Peyer's patches are swollen, hyperemic, and the seat of endothelial and lymphoid proliferation. They appear as flat, raised patches, of pinkish-gray color, somewhat soft in consistency, and with a smooth or rugous surface. During the second week more or less complete necrosis of the solitary and agminate follicles takes place—yellowish-green and greenish-brown sloughs (stained with bile) being formed. During the third week, by a process of ulceration, the slough is gradually separated and a more or less characteristic ulcer results. These ulcers are ovoid in shape (Peyer's patches), their long axis being in the direction of the long axis of the bowel; roundish ulcers (solitary follicles) are also quite frequently seen. The edge of the ulcer is undermined, its floor is smooth and made up of the muscular coat of the intestine or of the peritoneum. In some cases the latter is involved in the necrotic process and eventually becomes perforated. During the fourth week cicatrization ensues; it may be complete within a week or ten days, or it may be unduly protracted and become chronic. Often side by side one sees lesions in different stages of progress. Sometimes the cicatrices are visible for years—as a thinning of the mucous membrane, absence of the intestinal villi, and the presence of pigment.

These lesions while most common in the lymphoid tissues of the intestine, especially of the lower part of the ileum, occur throughout the body. The incidence of occurrence in different parts of the body is well exemplified by the following table, compiled by Baer, which gives in percentages the results of an analysis of the sites of ulcerations in over 1400 typhoid-fever necropsies: Small intestine (any part), 97.5; ileum, 89; ileum alone, 53.3; ileum (lower half), 36.8; large intestine, 33.3; cecum, 30.3; colon (not specific), 29.1; larynx, 19.9; ascending colon, 10.3; sigmoid flexure, 6.9; ileocecal valve, 6.7; jejunum, 5.9; large intestine, alone, 5.6; transverse colon, 4.5; œsophagus,4.5; descending colon, 2.6; vermiform appendix, 2.5; rectum, 2.4; pharynx, 2.2; cases without ulceration, but proved bacteriologically, 2.2; stomach, 1.98; Meckel's diverticulum, 1.1; trachea, 0.38; duodenum, 0.3; common bile duct, 0.23. In some cases the intestinal lesions are widespread and extensive; in other cases they are so slight as to be scarcely detectable; in some cases they are altogether absent—typhoid fever without intestinal lesions (typhoid bacteremia or septicemia).

The mesenteric lymph nodes, especially those corresponding to the portion of intestine affected, become hyperemic and swollen, largely in consequence of endothelial and lymphoid proliferation. Generally resolution takes place, but occasionally they become softened and necrotic, and may rupture into the peritoneum, causing peritonitis, or they may become caseous, calcareous, or atrophic. The lymph nodes throughout the body sometimes are swollen, and the superficial nodes may be palpable. The spleen becomes markedly enlarged (two or three times its normal size), its capsule tense, the pulp dark, reddish brown in color, and almost diffluent, and the Malpighian bodies

prominent. Usually it returns to its normal condition, but infarction and abscess formation may ensue.

Our knowledge of the nature of the foregoing lesions has been much enhanced by the studies of Mallory, who states that the essential lesion in typhoid fever is a diffuse proliferation of the endothelial cells giving rise to large epitheloid cells characterized by the possession of marked phagocytic properties, and that while this proliferation is most marked in the lymphoid tissue of the intestine, the mesenteric lymph nodes, the spleen, the bone marrow, and the liver, it may be found also to a varying extent in all the lymphatic vessels of the body, as, for instance, the lymphatics of the heart, lungs, testicles, pia-arachnoid, etc. The cause of these proliferative changes is a mild, diffusible toxin (an endotoxin, liberated upon disintegration of the typhoid bacilli) which circulates in the lymphatic and the blood stream. The swelling of the lymphoid nodes of the intestine, the mesenteric nodes, and the spleen is due almost entirely to these phagocytic cells, and the necrosis is purely accidental, resulting from occlusion of the veins and capillaries by fibrinous thrombi the consequence of degeneration of phagocytic cells beneath the lining endothelium of the vessels. These phagocytic cells enter the general circulation, passing through the liver and the lungs, on the one hand, and through the thoracic duct, on the other. Focal collections of these cells are found in different organs, the liver, the kidneys, the heart, the testicles, where they obstruct either the capillaries, the veins, or the lymphatics. Certain noteworthy changes occur in the bone marrow, as has recently been pointed out by Longcope: lymphoid proliferation, large phagocytes, foci of necrosis, more or less hyperplasia of the blood-forming cells, and, in the fatal cases, diffuse degenerative changes of the blood-forming cells, associated with marked œdema and congestion. Longcope suggests that these changes may be in some way related to, and perhaps responsible for, the leukopenia of typhoid fever.

In addition to the foregoing essential and characteristic lesions of typhoid fever, others often occur—the result of long-continued fever and toxemia and of secondary infection with pyococci—such as parenchymatous degeneration of the liver, kidneys, heart, voluntary muscles (Zenker's degeneration, especially of the abdominal muscles and the adductors of the thigh), etc. A mild grade of interstitial hepatitis sometimes develops; the so-called lymphomatous or acute interstitial nonsuppurative nephritis may occur; the gall-bladder may become inflamed, ulcerated, and even perforated (usually infection by way of the portal circulation); laryngeal ulceration may progress to and cause ulceration of the cartilages and perichondritis; hypostatic congestion of the lungs, and lobular or lobar pneumonia or pleuritis may supervene; cystitis and pyelitis, due to typhoid bacilluria, may arise; and thrombosis, especially of the veins of the lower extremity, said by Mallory to be due to the same sort of lesions that cause occlusion of the vessels in the intestine, may occur. In addition, abscesses in different parts of the body, peri-

ostitis, peripheral neuritis, meningitis, orchitis, parotitis, etc., have been observed. These, though often the consequence of mixed infection with pyococci, are sometimes due to the typhoid bacillus alone, and, as for instance in periostitis, they may remain dormant for a long time—months and years.

In the living subject the typhoid bacilli may be found in the stools, especially from the end of the first to the middle of the third week, during some relapses, and sometimes for months or years after an attack of typhoid fever (so-called typhoid carriers); in the urine, especially in cases of albuminuria, sometimes for long periods, even months, after convalescence; in the gall-bladder, during and for months or years after an attack of typhoid fever (the usual habitat of the organisms in the chronic typhoid carriers); in the blood from the spleen, the rose spots, and the peripheral circulation; sometimes in the sputum; in the cerebrospinal fluid in the event of certain complications, etc. In many of the complications and sequels, especially in post-typhoidal abscesses of the bones, subcutaneous tissues, etc., the bacilli may also be found, but they are usually associated with different pyococci. After death the typhoid bacilli have been found in the intestine, Peyer's patches, mesenteric lymph nodes, spleen, liver, kidneys, meninges, myocardium, bone marrow, blood, lymph, and divers complicating lesions.

Symptoms.—The period of incubation varies, as a rule, between ten and fourteen days, but it may be as short as five, or as long as twenty-one or more days. Occasionally this period is unannounced by symptoms. Usually, however, there are prodromes, such as malaise that increases with the onset of the febrile stage, headache, insomnia, anorexia, sometimes diarrhœa, and, especially in children, vomiting. In most cases the onset of the disease is insidious, the patient complaining of gradually increasing malaise, disinclination to exertion, headache, neuromuscular pains in the back and limbs, anorexia, epistaxis, possibly chilly sensations and feverishness; finally, he takes to bed—from which day we date, though often incorrectly, the onset of the disease. Occasionally the onset is sudden and manifested by severe headache, pains in the back and legs, chills that may be repeated, marked prostration, etc.

During the first week the symptoms mentioned become aggravated; the temperature rises in a characteristic step-ladder manner; the tongue, though it remains moist, becomes furred; the pulse becomes increased in frequency (90 to 100); there is thirst; the abdomen becomes distended and tender on pressure; though there may be diarrhœa, constipation is the more likely; and there may be slight cough due to bronchitis.

At the end of the first week, sometimes by the fourth or fifth day, the fastigium is reached. All of the symptoms become aggravated; the temperature reaches its highest point (usually 103° to 104° F.), and it remains high, the daily remissions being less than during the first week; the pulse becomes accelerated, 90 to 110, and dicrotic (an early feature); the headache gives way to mental hebetude and later to delirium, especially at night; the patient is often quite deaf; and the typhoid state

frequently supervenes. The skin is usually hot and dry, but frequent attacks of profuse perspiration, followed by sudamina, may occur. About the eighth day of the disease a number of rose-colored spots, that are quite pathognomonic, appear on the abdomen, the chest, and frequently elsewhere, especially on the back. The spleen is enlarged, distinctly palpable, and somewhat tender. The tongue becomes dry and later fissured, and sordes may form on the teeth and gums. The abdomen may become markedly distended, gurgling in the right iliac fossa may develop, and the constipation may give way to diarrhœa. The stools are of a pale yellow (pea-soup) color and highly malodorous (characteristic typhoid stool). The fastigium usually lasts from ten days to two weeks.

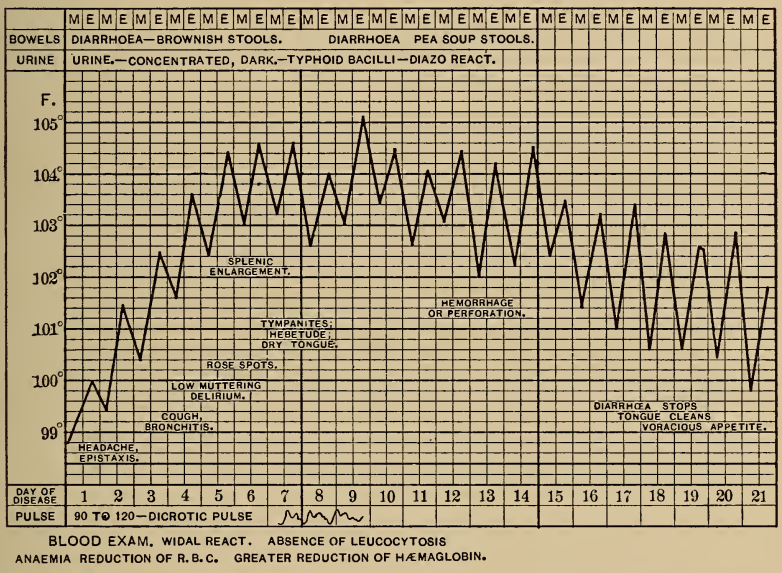
Toward the end of the fastigium, that is, in the third week, the typhoid state becomes more pronounced, and the pulse more rapid, 110 to 130 or more; the temperature, previously continuous, becomes remittent, and usually somewhat lower than during the second week; and any of the many complications may arise. In favorable cases at about the twenty-first day of the disease (the commencement of the fourth week) the stage of decline or defervescence sets in. The fever lessens and finally becomes intermittent with less and less diurnal variations; the local and general symptoms abate; and convalescence ensues.

From the foregoing, which represents in outline the course of an ordinary or average case of typhoid fever, there are many departures; the symptoms vary much with the virulence of the infecting microörganisms, the resistance of the individual, and the presence or absence of complications and secondary infections—due usually to pyogenic cocci.

The Fever.—In the majority of cases, during the first four or five days of the disease, a daily increase of temperature of 1.5° to 2° F., and a morning remission of from 1° to 1.5° F., occur—producing what is spoken of as the step-ladder or zig-zag course of the fever (Fig. 1). The temperature usually reaches its maximum on the fourth, fifth, or sixth day. Then follows the fastigium, which lasts usually from ten to fourteen days, and during which the temperature is more uniform and of the continuous type, the morning temperature being 102° to 103° F., and the evening from 103° to 104° F., or higher. In ordinary cases toward the end of the fastigium (thirteenth to sixteenth day) the temperature becomes remittent, and the evening rise shows some tendency to be less marked. At the end of the fastigium (sixteenth to twenty-first day) the temperature is usually intermittent; the morning temperature approaching if not quite reaching the normal, and the evening temperature being 101° to 101.5° or 102° F. Gradually the evening temperature also becomes normal. In cases of moderate severity the temperature may begin to decline at the end of the second week and soon reach normal; in severe cases it may remain high (104° to 105° F.) in the evening throughout the third week, even into the fourth week, and then gradually decline. Defervescence occurs usually by lysis, rarely by slow crisis.

Variations in the course of the temperature, however, are quite common. Thus, the early step-ladder rise may be missed. During the greater part of the fastigium the temperature may be 105° to 106° F. or more (hyperpyrexia). Even higher temperatures are occasionally met with—107°, 109°, 110° F., especially in the event of certain complications, such as pneumonia, septic manifestations, pyococcic infections, etc. Again, the fever may be unduly protracted, continuing four, five, six, or more weeks—usually the consequence of certain inflammatory complications (secondary infections), such as suppurating mesenteric lymph nodes, or sluggish intestinal ulcers, post-typhoidal anemia, intestinal auto-intoxication, etc. Sometimes the

FIG. 1


M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E
BOWELS
DIARRHOEA—BROWNISH STOOLS.
DIARRHOEA PEA SOUP STOOLS.
URINE
URINE.—CONCENTRATED, DARK.—TYPHOID BACILLI—DIAZO REACT.
F.
105°
104°
103°
102°
101°
100°
99°
SPLENIC ENLARGEMENT.
HEMORRHAGE OR PERFORATION.
TYMPANITES; HEBETUDE; DRY TONGUE.
ROSE SPOTS.
LOW MUTTERING DELIRIUM.
COUGH, BRONCHITIS.
HEADACHE, EPISTAXIS.
DIARRHŒA STOPS TONGUE CLEANS VORACIOUS APPETITE.
DAY OF DISEASE
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21
PULSE
90 TO 120—DICROTIC PULSE
BLOOD EXAM. WIDAL REACT. ABSENCE OF LEUCOCYTOSIS
ANAEMIA REDUCTION OF R. B. C. GREATER REDUCTION OF HÆMAGLOBIN.

The temperature curve and the chief symptoms in typhoid fever. (Musser.)

temperature is distinctly intermittent throughout the course of the disease, even in the absence of complicating malaria. In other cases the fever may be slight throughout the entire course, or the temperature may be scarcely above normal (apyrexial form), or even subnormal. It is quite often subnormal during convalescence.

Sudden temporary falls of temperature are sometimes met. They are due to: (1) Intestinal hemorrhage; (2) intestinal perforation; (3) cardiac failure or vasomotor collapse; (4) abortion or premature labor; (5) the use of coal-tar products; (6) ill-understood nervous factors; and (7) they sometimes occur without obvious cause.

Post-typhoidal elevations of temperature (recrudescences) are quite common. They sometimes occur during the period of defervescence,

but more frequently during convalescence—after the morning and evening temperatures have been normal for several days. The recrudescence often occurs rather suddenly, the temperature may reach a high grade (103° to 105° F.), but usually it subsides within three or four days, although it may last a week. Such recrudescences are attributable to dietetic errors, constipation (auto-intoxication), emotion and excitement attending the visits of too solicitous friends, etc. If protracted the fever is significant of some complication, or of anemia, or it may be what has been aptly termed "bed fever"—due to low diet and too prolonged stay in bed.

Chills, according to Osler, occur (*a*) sometimes with the fever of onset; (*b*) occasionally at intervals throughout the course of the disease, and followed by sweats (so-called sudoral form); (*c*) with the advent of complications, pleurisy, pneumonia, otitis media, periostitis, etc.; (*d*) with active antipyretic treatment by the coal-tar products; (*e*) occasionally during the period of defervescence without relation to any complication or sequel, probably due to septic infection; (*f*) according to Herringham, chills may result from constipation; there are some cases in which during the latter half of the disease chills recur with great severity.

Skin.—In from 75 to 85 per cent. of all cases a characteristic roseolous rash appears on the body on from the seventh to the ninth day; rarely it may be delayed to the twelfth day. It consists of isolated, circular, slightly elevated macules or maculo-papules, from 2 to 4 mm. in diameter. They are found almost always on the trunk, especially on the abdomen and the lower chest; but their absence should not be certified to until the back has been thoroughly examined. Sometimes they are present on the arms and the legs, and the face; occasionally in large numbers. They disappear upon pressure and reappear immediately the pressure is removed. Usually they come out in successive crops, each crop consisting, as a rule, of from six to a dozen or more spots. Each spot lasts about four or five days, so that fading spots are seen side by side with others of a newer crop. They continue to appear until the end of the second week; sometimes even in the third week, and occasionally during convalescence; and they are not infrequently observed during a true relapse. In some cases they are entirely absent; in other cases they are numerous and cover the entire body. When numerous they are usually darker than when fewer; occasionally they are hemorrhagic. A fine, furfuraceous desquamation is not infrequent; occasionally the desquamation is flaky.

Other eruptions are often encountered. Thus sudamina and miliaria are common, especially during the second and the third weeks; they are usually associated with profuse perspiration and not infrequently with chills—sudoral typhoid, of the French. A diffuse scarlatiniform erythematous rash (in some cases undoubtedly a drug eruption) has sometimes been observed, especially during the first week. Morbilliform rashes, urticaria, and herpes are occasionally seen. Rarely one encounters, especially on the abdomen, small bluish spots that do not

disappear on pressure (maculæ ceruleæ, *taches bleuâtres* of Trousseau, or peliomas); they have been attributed to, but are probably not always due to, pediculi. Again, petechiæ and purpuric spots occur, especially in debilitated subjects; and rarely even extensive gangrenous dermatitis (due, doubtless, to infection of the skin), of which I have seen a number of cases. More or less extensive furunculosis is not uncommon. Bed sores are not so common as formerly—due to improved nursing; but they may occur in debilitated subjects, especially if the disease be protracted. Loss of the hair and cessation of the growth of the nails are not uncommon. Œdema of the skin and subcutaneous tissues may result from venous thrombosis, anemia, or nephritis.

Digestive System.—The tongue is usually coated throughout the course of the disease; at first moist, it soon becomes dry, swollen, brown, and fissured. I have seen severe acute glossitis. The gums are not infrequently covered with sordes (epithelial debris, food particles, bacteria, etc.). Stomatitis progressing to hemorrhage and ulceration may ensue. Parotitis, unilateral or bilateral, and due to extension of infection along Stenson's duct or rarely to metastatic pyogenic infection, is occasionally observed. Extension of infection along the Eustachian tube may result in suppurative median otitis. Cancrum oris is rare; I have seen a case from which the diphtheria bacillus was isolated. Sore throat is sometimes complained of; the tonsils may be swollen and congested, and present whitish spots of lacunar or other inflammation which may proceed to ulceration (tonsillar typhoid). Catarrhal and ulcerative pharyngitis and œsophagitis are by no means infrequent, and may be followed by stenosis. Anorexia is the rule. Nausea and vomiting are unusual, although they may be present at the outset, and have been known to recur about the third week or to continue throughout the course of the disease, and to contribute to the fatal issue. The gastric juice is deficient in amount, and hydrochloric acid is usually much diminished or absent. While diarrhœa may be present at the beginning and continue throughout the course of the disease, constipation is the rule at the onset and not infrequently persists. Usually, however, at about the end of the second week, if not earlier, diarrhœa (four to ten or more stools in the twenty-four hours) appears; but even marked constipation may be present with even extensive ulceration of the ileum. The diarrhœic stools are quite characteristic: of the color and consistency of pea-soup, alkaline, and very malodorous; usually they separate on standing into an upper turbid liquid layer, and a lower thick sedimentary layer, consisting of partly digested food, desquamated, epithelial cells, necrotic-tissue elements, blood corpuscles, granular debris, triple phosphate crystals, and numerous bacteria. Cultures may reveal the typhoid bacillus.

Tympanites is common, but rarely nowadays so marked as to be grave. When marked, by interfering with the action of the diaphragm, it occasions dyspnœa, and may embarrass the heart, and also favor intestinal perforation. Gurgling and pain on pressure may be found

in the right iliac fossa. Spontaneous pain in the abdomen is present in about one-third of the patients, and contrary to former opinions according to which it was believed always to indicate or suggest perforation and peritonitis, it may be due to a wide variety of diseased conditions: it is not infrequently due to extra-abdominal conditions, such as pleurisy or pneumonia, or even femoral phlebitis; it is a not infrequent concomitant of an enlarged spleen, of a distended urinary bladder, of severe diarrhœa or marked constipation, and sometimes it attends intestinal hemorrhage. Usually, however, it suggests serious complications, of which the most important are intestinal perforation, appendicitis, and cholecystitis.

Intestinal hemorrhage occurs in from 3 to 7 per cent. of the cases. It is rarer in children. It may occur at the end of the first week, when it is usually slight and due to marked congestion; usually it occurs toward the end of the second week, or during the third week, or later. The hemorrhage may occur once or repeatedly; and the amount of blood lost may be so slight as almost if not quite to elude detection, or so extreme as to lead immediately to a fatal termination (a rather unusual event). Slight hemorrhages are unattended by subjective symptoms; large hemorrhages are indicated by sudden and rapid fall of the temperature (2° to 8° F.), pallor, a sensation of faintness, a rapid and small pulse, low and falling blood pressure, extreme prostration, and visible blood in the stools (though even death may occur without the discharge of blood from the bowel). Clinically, a single, even rather large, hemorrhage is not necessarily a serious phenomenon, since patients often improve after a hemorrhage, especially when it occurs early; but repeated hemorrhages are likely to lead to a fatal termination.

Perforation occurs in from 3 to 4 per cent. of the cases, and is responsible for one-fourth or more of the deaths. It bears no direct relationship to the severity of the infection, since it is not infrequent in mild cases, although most common in the severer infections, especially those attended by tympanites and hemorrhage. It is more common in males than in females, and is rare in children. Although it may occur at any time during the course of the disease, even during the first week as well as very late during protracted convalescence or relapses, at least one-half of the cases occur during the second and the third weeks (most commonly in the third week). The perforation involves the ileum in 88 per cent. of the cases, the colon in 4.7 per cent., and the vermiform appendix in 4.7 per cent. (Scott).

The symptoms of the perforation are those of the rupture (which rarely occurs almost if not quite unannounced) and those of the consecutive peritonitis (which I have several times seen at necropsy without noteworthy clinical signs). The most important symptom of the perforation is pain (scarcely missed unless the patient is comatose), which usually comes on suddenly, is sharp and paroxysmal in character, commonly situated in the right lower abdominal quadrant, and not infrequently increases in severity for some time. Concurrent with the pain

there are often the usual evidences of collapse (fall of the temperature often to or below the normal, increased frequency and smallness of the pulse, vomiting, pallor, perspiration, etc.), and subsequently the ordinary manifestations of peritonitis. These comprise local pain, tenderness, and rigidity of the abdominal muscles, and, as a rule, increasing abdominal distention and limitation of the respiratory movements. Replacement of the liver flatness by tympany is of value only when it develops rapidly and is present in a flat or slightly distended abdomen, and when by changing the posture of the patient flatness and tympany alternate; even marked tympany may be due merely to intestinal distention. Peritoneal exudate may occasion dulness in the flanks, which may shift its position with change in the posture of the patient, and may even be recognizable by rectal examination. Auscultation may reveal peritoneal frictions, sometimes within a few hours; later there may be an entire absence of peristaltic sounds and movements. The general signs of peritonitis are often relatively inconspicuous as contrasted with the local signs. As a rule, however, the Hippocratic facies, pallid, dusky, pinched, and clammy, is present; the temperature soon rises (after the initial fall) and becomes intermittent; the pulse-rate and the respiration-rate become increased; and hiccough and vomiting sometimes develop. Leukocytosis is a valuable, though inconstant, sign.

Peritonitis may be due also to infection passing through an ulcer that has not perforated; to gangrene of the intestine; to softening and rupture of a mesenteric or retroperitoneal lymph node; to splenic infarction and abscess; to a twisted and necrotic Meckel's diverticulum; to cholecystitis, appendicitis, salpingitis, etc. The swollen mesenteric lymph nodes are sometimes palpable through the abdominal wall.

The liver is often enlarged from cloudy swelling, and is occasionally somewhat tender. Jaundice is occasionally observed; it may be a toxic process or due to cholangitis and obstruction. Cholecystitis is quite common, and may occur during the course of the disease or during convalescence—when the local signs being in abeyance the condition is not infrequently thought to be a "relapse." The symptoms, as a rule, however, consist of local pain, tenderness, and muscular rigidity, and a palpably enlarged gall-bladder; vomiting sometimes occurs at the onset; jaundice is inconstant. Perforation of the gall-bladder is revealed by pain, fall of temperature, collapse, and consecutive peritonitis. Typhoid bacilli are often found in the gall-bladder after death in the absence of local disease, and it is to such latent biliary infection that many of the cases of true relapse are attributable. Cholelithiasis not infrequently follows typhoid fever, and from the gallstones the typhoid bacilli have been cultivated. I have observed several cases of primary typhoid cholecystitis (recovery of the typhoid bacillus) without other evidences of typhoid infection. Suppurative cholangitis, suppurative pylephlebitis, and liver abscess have occasionally been observed.

Respiratory System.—Epistaxis is a significant symptom of the early stages of typhoid fever, and occurs usually at the end of the first and the

beginning of the second week. A simple laryngitis is not very uncommon. Œdema, ulceration, perichondritis, and paralysis of the larynx are quite rare. Bronchitis is a part of the disease, and one of the early manifestations. It is usually slight or moderate in degree only, and revealed by cough, slight expectoration, harsh vesicular breath sounds, and a few sibilant and fine bubbling rales. Rarely the finer tubes are involved—whence bronchopneumonia may supervene. Bronchopneumonia may also follow œdema and congestion of the lungs, which are by no means uncommon during the second and third weeks; during the same periods, when the patient is more or less stuporous, an aspiration pneumonia is not infrequent. True croupous pneumonia may also occur during the later half of the disease, and is a serious complication. Occasionally it is an initial manifestation—pneumotyphoid (pneumonia due to the typhoid bacillus, with typhoid bacilli in the sputum); in this event the early signs are those of pneumonia, the intestinal and other manifestations of typhoid fever only developing later. Pulmonary abscess or gangrene and pneumothorax are rare sequels. Pleurisy may be primary or secondary: secondary to any of the aforementioned pulmonary conditions; primary and due to direct infection with the typhoid bacillus —pleurotyphoid. Occurring early, the lesions are usually dry or serous, later often purulent. The bronchial lymph nodes have been known to soften and suppurate.

Circulatory System.—The pulse is usually increased in frequency, though not proportionately to the fever. During the first week it is less than 100, as a rule; in the second week it commonly fluctuates below 110 and becomes dicrotic (an early and quite characteristic feature). With the progress of the disease the evidences of vasomotor paresis become manifest—low blood pressure, increased frequency of the pulse (130 or more sometimes), which often becomes running or thready, capillary stasis (coldness and lividity of the extremities), overfilling of the splanchnic vessels, anemia of the cerebral vessels, and sometimes vasomotor collapse. As convalescence sets in vasomotor tone is restored, and the pulse becomes slower, fuller, and stronger; during convalescence the pulse is often unusually slow (a characteristic feature). Changes in the heart muscle (parenchymatous degeneration, and acute interstitial myocarditis) are common. Often the only noteworthy clinical manifestation is feebleness of the first sound of the heart. When more marked, however, there is not infrequently gallop rhythm, or embryocardia, or dilatation of the heart (systolic murmur, increase of the dulness, etc.). Endocarditis and pericarditis are uncommon, but not unknown.

Venous thrombosis, especially of the lower extremity (femoral, popliteal, saphenous, or superficial veins), is not infrequent, particularly during convalescence. It may be due to thrombophlebitis (in which event the typhoid bacillus may be cultivated from the vein and the clot), or to cardiac weakness and slowing of the circulation; the more common occurrence of the thrombosis in the left leg may be due to the pressure exerted by the right iliac artery crossing the left

iliac vein, as well as to congenital adhesions, which are ten times as common in the left as in the right iliac vein. The thrombosis is evidenced by swelling and œdema of the leg, and by pain and tenderness along the course of the involved vein. The process usually subsides without ill effects, but it may require a long time, and embolism of the pulmonary artery, ensuing on dislodgement of the thrombus, has been known to result in death. Infarcts of the internal organs are not very uncommon, and may result from arterial thrombosis or embolism. Gangrene sometimes results from occlusion of one of the larger arteries, and a number of cases of more or less local (though sometimes widespread) gangrene unrelated to definite vascular distribution have been reported; they are probably due to secondary infection of the skin (gangrenous dermatitis). Heart clot is rather unusual.

Blood and Hemopoietic System.—Leukopenia is present throughout the disease, except in the event of complications, although a temporary increase in the leukocytes in the peripheral circulation is not infrequently produced by the cold baths. With the leukopenia there is a disproportionate increase of the large mononuclear leukocytes and a reduction of the polynuclear neutrophiles (to 60 per cent. or less). An increase of the polynuclear neutrophiles is often an early manifestation of inflammatory complications (perforation, cholecystitis, etc.) and may be present with little or no increase in the total number of leukocytes. The blood serum agglutinates the typhoid bacillus (Gruber-Widal reaction). During the later half of the disease a rather well-marked anemia usually develops; during convalescence this may assume a high grade and constitute a serious feature (post-typhoidal anemia). The spleen is enlarged and palpable in at least three-fourths of the cases. It may extend two, three, or more finger breadths beyond the free margin of the ribs, and it is not infrequently quite tender. The enlargement is sometimes missed in elderly subjects and in the event of perisplenitis. I have seen infarction and abscess of the spleen; rupture has been reported. The superficial lymph nodes throughout the body are sometimes palpably enlarged.

Nervous System.—Persistent headache (usually temporal and occipital) is a prominent and significant symptom of the first week, and is often associated with severe pains and aching in the back and legs. During this period also restlessness and insomnia are likely to be present, and are often distressing. Sometimes the patient is quite deaf. The pupils are usually markedly dilated. Gradually, in most cases, as the patient succumbs to the toxemia, that is, during the second week, the headache, restlessness, and insomnia are replaced by mental dulness, hebetude, and delirium, which vary considerably in different cases, and in the beginning at least are usually most marked at night. The patient becomes listless and apathetic, and is more or less unconscious of his surroundings, although he may lie for hours at a time with his eyes wide open (coma vigil); he frequently mutters constantly, picks at the bedclothing or at imaginary objects (carphologia), and exhibits general

muscular twitchings, especially of the tongue, lips, hands, and fingers (subsultus tendinum). Often the undue muscular irritability is quite manifest by the ready production of myoidema. In this, the typhoid state, the urine and feces may be voided involuntarily, and the patient knows not even his wants. Not infrequently he shows a disposition to get out of bed—whence he should not be left alone for even an instant, as not a few patients have thrown themselves from a window during the temporary, though only momentary, absence of the nurse. Occasionally, instead of a quiet, muttering delirium, the patient may exhibit a noisy, hysterical (emotional), or maniacal delirium (especially alcoholic subjects, in whom the condition quite resembles delirium tremens).

Occasionally typhoid fever is ushered in with unusual nervous symptoms. The headache and backache may be intense, and there may be also photophobia, pain, tenderness, and rigidity of the muscles of the neck, sometimes even retraction of the head, and rarely convulsions (more common in children than in adults). In other cases, later in the course of the disease, similar symptoms arise. These cases suggest and are only with difficulty distinguished from meningitis; three types may be distinguished: (1) Cases suggesting meningitis, but in which no anatomical lesions can be demonstrated—the so-called meningism of the French, probably due solely to toxemia; (2) cases exhibiting the lesions of serous meningitis and perhaps revealing the typhoid bacillus in the cerebrospinal fluid; and (3) cases of purulent meningitis from which the typhoid bacillus, either alone or in association with other bacteria, may be isolated. Circumscribed encephalitis, and hemorrhage, embolism, and thrombosis of the brain (hemiplegia with or without aphasia) have been observed. Myelitis or poliomyelitis (spastic or flaccid paralysis) is still more uncommon. Peripheral neuritis is quite frequently observed, of which a well-known manifestation is the rather common tender toes and tender feet—believed to be more common since the use of the cold-bath treatment. The very common painful calves (observed frequently during convalescence), as well as the much less common painful muscles in other parts of the body, are not infrequently due to neuritis; but they are sometimes due to myositis or to thrombosis of a vein. Post-typhoidal insanity (confusional insanity, melancholia, or mere mental dulness) is not uncommon. It is usually attributed to toxemia and impaired nutrition, and in the majority of cases soon subsides. Oculomotor paralysis, paralysis of accommodation, iritis, keratitis, conjunctivitis, and divers other ocular complications have been reported.

Genito-urinary System.—The urine, as a rule, presents the features of so-called febrile urine (diminished in amount, high colored, of high specific gravity, abundance of urates, etc.). The diazo reaction is usually present. At least three-fourths of the patients exhibit slight albuminuria—attributable to toxic degeneration of the renal epithelium. Occasionally a true nephritis (hemorrhagic or non-hemorrhagic) occurs at the height of the disease. I have observed acute interstitial non-suppurative nephritis (the so-called lymphomatous nephritis of Wagner).

Nephritis during convalescence or as a sequel is quite rare, the lesions developing during the course of the disease usually subsiding in the non-fatal cases. Retention of urine is quite common and often occasions rather severe abdominal pain. Typhoid bacilluria is present in at least one-third of the cases, and sometimes lasts for months after apparent recovery from the disease; because the urine is often not disinfected this bacilluria is answerable for some epidemics. The bacilluria may be due to pyelitis, cystitis, spermatocystitis, prostatitis, or orchitis, or it may occur without noteworthy lesions. Diabetes (or glycosuria), hematuria, and hemoglobinuria are rarely observed. Menstruation is not infrequent at the beginning of the disease, and pregnant women usually abort.

Other unusual complications comprise osteomyelitis, periostitis, perichondritis, synovitis, and arthritis (from which the typhoid bacillus may be cultivated months and even years after the primary infection). Occasionally during or after convalescence the patient complains of pain, tenderness, and rigidity in the lumbar and sacral regions, and of certain nervous (sometimes hysterical) phenomena. This condition (so-called typhoid spine) is in some cases a pure neurosis, but it not infrequently has an organic basis—spondylitis or perispondylitis. Perinephritic abscess may simulate the condition. Fever may be an important sign in the differential diagnosis.

Typhoid fever is not uncommonly associated with other diseases, especially tuberculosis, malaria, measles, diphtheria, noma, erysipelas, furunculosis, etc. Most of the cases of so-called typho-malarial fever are true typhoid fever; others are remittent malarial fever; in a few cases only do two diseases co-exist.

Varieties of Typhoid Fever.—Many different varieties of typhoid fever have been described; but the infection is always with the same typhoid bacillus (which, however, often exhibits variations in certain of its biological characteristics); the clinical differences are dependent, therefore, upon differences in the virulence of the infecting agent, upon unusual localizations of the infection, and upon variations in the resistance of the individual. Thus, mild (typhus lævis sive lævissimus, Griesinger) and severe forms are readily distinguished. In the mild forms, which are frequently overlooked and especially common in children, the clinical and anatomical manifestations of the disease are ill developed, and although fever, an enlarged spleen, rose-spots, the Gruber-Widal reaction, etc., often occur, convalescence is not infrequently established at or before the end of the second week; but the disease may run the more usual course of three to four weeks. In children the abdominal symptoms are often slight or absent, whereas the nervous, cerebral symptoms are likely to be marked.

Unusually acute and rapid cases are sometimes observed. Doubtless an abortive form occurs, but it is very uncommon—more especially since the general use of the Gruber-Widal reaction. As described, the onset presents nothing unusual, except in some cases rather sudden and marked initial fever; but at the end of the first week the fever falls, and

convalescence goes on as in the ordinary cases, though relapses may occur. Latent or ambulatory typhoid fever is a form of infection in which the symptoms, at the beginning at least, are so mild that the patient does not feel the necessity of going to bed, and therefore continues about until some unusual phenomenon, such as marked diarrhœa, delirium, intestinal perforation, or hemorrhage, etc., brings him under the attention of the physician. The subsequent course of the disease is often severe and not infrequently fatal. The initial symptoms of typhoid fever sometimes suggest an ordinary "cold," influenza, tonsillitis, or gastritis. An afebrile, exceedingly rare, form of the infection has been described by Liebermeister. The severe cases are characterized, as a rule, by unusual severity of the fever and of the nervous symptoms (delirium, prostration, vasomotor paralysis, etc.)—whence these are sometimes spoken of as the cerebrospinal form. In other cases there is unusual localization of the lesions in different organs—whence the terms pneumo-typhoid, pleuro-typhoid, nephro-typhoid, tonsillo-typhoid, pharyngo-typhoid, meningo-typhoid, etc. A few severe cases have been characterized by petechial spots and hemorrhages from the mucous membranes—so-called hemorrhagic form. The disease often runs a severe course in elderly, alcoholic, and obese subjects.

Relapses.—A relapse, a repetition of the anatomical and clinical features of the diease and due to re-infection, is not an uncommon event, occurring in different epidemics in from 3 to 15 per cent. of the cases. It should be distinguished from recrudescence of the fever (spurious relapse, previously alluded to), as well as from complications, such as cholecystitis, pleuritis, etc., for which it is frequently mistaken. It is not always possible to discover the source of re-infection: sometimes it may be from without; usually it is from within, and one should always bear in mind the possibility of infection from the gall-bladder which frequently harbors typhoid bacilli without obvious local disease; the urinary tract also may be the source of reinfection; but the real cause of the relapse is most likely to be found amongst the problems of immunity—perhaps in temporary exhaustion or overpowering of the bacteriolytic activities of the blood. As a rule, the relapse comes on within seven days of the beginning of the convalescence; sometimes it develops earlier, even before the temperature has reached normal (intercurrent relapse), in which event it is often severe and protracted; occasionally it may not come on until the temperature has been normal for three weeks or more. The period of apyrexia is usually without noteworthy manifestations. The onset of the relapse, as a rule, is sudden, the temperature rising abruptly, sometimes with a chill or chilliness. Then follow usually the characteristic step-ladder rise of the temperature (for two or three days), new roseolous spots (on the second to the fourth day), and enlargement of the spleen—two of which signs, at least, should be present to warrant the diagnosis of a true relapse. As a rule, the relapse does not last more than ten or fourteen days, and the clinical signs are less severe than in the primary attack;

but occasionally all the phenomena of the disease are much aggravated during the relapse, and the patient may die of the toxemia or of one of the common accidents, such as hemorrhage or perforation. There may be a second, a third, a fourth, and even a fifth relapse. A recurrence, as contrasted with a relapse, means another attack of the disease occurring after complete restoration to health. Usually, typhoid fever confers lasting immunity, but second and apparently well-authenticated third attacks have been observed—a period of years intervening between the attacks.

Diagnosis.—The recognition of typhoid fever is, in the one case, a matter of the greatest ease, and in another, of the greatest uncertainty —for a time at least. No one symptom is conclusive. Of special value in the diagnosis are prodromal symptoms lasting for a week or more, the gradual onset of fever (of characteristic rise, if it has been observed), persistent headache, nose-bleed, roseolous spots, enlarged spleen, early dicrotism of the pulse, leukopenia, and the diazo reaction; the diagnosis is made conclusive by the Gruber-Widal reaction (in dilutions of 1 to 50 or more), and the cultivation of typhoid bacilli from the blood, the rose-spots, the stools, the urine, or the cerebrospinal fluid. In some regions continued fever for seven days without evidence of local disease is quite suggestive. Paratyphoid fever, as a rule, is first suggested by the absence of the Gruber-Widal reaction with the typhoid bacilli. It may be difficult or impossible for some days to exclude cerebrospinal meningitis, since typhoid fever sometimes begins with severe nervous manifestations, but the characteristic course of the fever, the erythematous rose rash (as contrasted with a petechial eruption), the presence of abdominal symptoms, the absence of herpes facialis, the presence of the Gruber-Widal reaction, and the negative results of lumbar puncture, serve ultimately to distinguish typhoid fever. The continued types of malarial fever, which much resemble typhoid fever, may be distinguished by the absence of the Gruber-Widal reaction, the presence of the malarial parasite in the blood, and by the administration of quinine. Acute miliary tuberculosis is often differentiated with difficulty, but it is suggested by absence of the characteristic fever curve, of abdominal symptoms, of erythematous rose-spots, of leukopenia, and of the Gruber-Widal reaction, and by the presence of increased pulse-rate, of dyspnœa, of cyanosis, of occasionally bloody expectoration, of choroidal tubercles, and of tubercle bacilli in the cerebrospinal fluid and rarely in the blood. Certain pyemic processes, of which ulcerative endocarditis is an example, are usually diagnosticated typhoid fever, but they may sometimes be differentiated by detecting a source of infection, and by the presence of undue dyspnœa, irregular heart action, a peculiar rough systolic, but more especially a diastolic murmur, irregular and repeated chills or chilly sensations, profuse sweats, the early occurrence of marked prostration, the rapid development of anemia, leukocytosis, embolic phenomena (petechial hemorrhages, infarctions, etc.), and pyococci in the blood, and by the absence of a characteristic fever curve, of persistent headache,

of epistaxis, of abdominal symptoms, and of the Gruber-Widal reaction. Acute leukemia without noteworthy enlargement of the lymph nodes, which usually resembles typhoid fever, may be distinguished by the absence of the Gruber-Widal reaction and the presence of leukocytosis (lymphocytosis). Pneumonia, appendicitis, and tuberculous peritonitis also must be considered in the differential diagnosis in certain instances.

Prognosis.—The prognosis is always doubtful, since no one can foretell whether or not one of the two accidents (hemorrhage and perforation), responsible for almost two-thirds of the deaths, will occur. The mortality varies much in different epidemics—from 5 to 12 per cent. in private practice to from 7 to 20 per cent. in hospital practice, where the worst cases are encountered. During recent years, in consequence of the general use of the cold-bath treatment, the mortality has certainly been reduced—in some hospital statistics to less than 4 per cent. The prognosis in general depends upon the severity of the infection, the resistance of the individual, and the presence or absence of complications. Aside from repeated hemorrhages and perforation (with consecutive peritonitis) answerable for almost two-thirds of the deaths, especially unfavorable symptoms are persistent high fever, persistently rapid pulse (above 120), profound toxemia and delirium, marked tympanites, and complications, such as pneumonia, meningism, meningitis, etc. Sudden death may occur during the fastigium, as well as during defervescence or convalescence, from cardiac collapse, vasomotor paralysis, pulmonary or cerebral embolism or thrombosis, etc. The passage of eighty to one hundred or more ounces of urine daily is one of the most favorable prognostic signs The prognosis of relapses is not essentially different from that of the primary attack. The prognosis in children, as a rule, is good.

Treatment.—In typhoid fever probably as much as, if not more than, in any other disease, our duty is not to our patient alone, but in a special sense to the community at large Since typhoid fever is a preventable disease, it should be prevented; the duty of preventing it falls upon the physician no less than upon the public health authorities. The general measures of prophylaxis set forth on page 29 should be scrupulously followed.

Whether Wright's preventive inoculation will ultimately commend itself to the profession is at least doubtful. According to British army surgeons, typhoid fever is more than two and a half times as common, and one and a half time as fatal, among the uninoculated as compared with the inoculated troops. Of possible value during times of war, inoculation is not likely soon to attain much vogue in civil practice; it may prove of service among hospital attendants. The serum treatment of the disease, for which Chantemesse and others claim good results, has not commended itself to the profession at large. A satisfactory vaccine (bacterin) treatment has not yet been devised.

Since typhoid fever is a self-limited infection, the special function of the physician is to assist, never thwart, the well-directed efforts of

nature. This comprises attention to, (1) the general management of the patient, (2) the diet, (3) hydrotherapy, and (4) medicinal and other measures indicated symptomatically.

General Management.—Whether the patient shall be treated in his home or in a hospital is a matter to be decided in each individual case; in general the hospital patient has the advantage, since the nursing, as a rule, is better and the necessary manipulations can be more easily carried out; disinfection is more thoroughly practised; complications are likely to be sooner recognized (since a physician is always at hand); and in the event of surgical intervention being necessary, it can be undertaken sooner and under better auspices than in the patient's home. But under proper supervision, the patient may be very well treated at his home. Under all circumstances, he should be isolated—in the hospital, in a ward or room set apart for this purpose; at home, in a room affording abundance of sunlight and fresh air. He should be confined absolutely to bed until he is well along in convalescence, and he must use a bed-pan and a urinal. Special attention should be directed to avoiding creases in the bed-linen so as to promote the comfort of the patient and prevent, as far as possible, the development of bed sores. The patient should be turned on his side from time to time, and his skin, especially of the back, should be frequently sponged with dilute alcohol (50 per cent.) or with equal parts of alcohol and alum water; following this a drying powder may be applied, such as zinc stearate, or equal parts of powdered zinc oxide and boric acid. Should the skin become irritated, one may substitute a mixture consisting of boric acid 10 grains (0.65 gram), zinc oxide 1 to 2 drams (4 to 8 grams), and castor oil 1 ounce (30 grams). The mouth and throat should be kept clean and sweet by the use of mild antiseptic solutions, such as boric acid (3 per cent.); or boric acid (1 dram, 4 grams), glycerin (6 drams, 25 grams), and rose water (6 ounces, 200 grams); or liquor antisepticus (U. S. P.); or the following:

℞—Carbolic acid	6 minims	0	40
Oil of eucalyptus	10 minims	0	60
Sodium borate	1 dram	4	00
Glycerin	3 drams	12	00
Distilled water, sufficient to make . . .	3 ounces	100	00.—M.

The physician should always write his orders in detail, rather than trust to the memory of even a well-trained nurse; in the absence of a trained nurse, this is all the more imperative.

The general management of convalescence often demands much tact on the part of the physician—to withstand the importunities of the patient and the usually well-meant, but often ill-advised, endeavors of his solicitous friends. Could solicitous friends be banished for a while, most typhoid-fever patients would fare better; at all events, the visits should be as short as possible, and all matters that may excite the patient

or disturb his emotions should be scrupulously avoided. On the fifth or the sixth day of normal temperature, in the ordinary case, the patient may be propped up in bed for a while, and on the eighth day or thereabouts he may sit in a chair. A general tonic is usually advantageous, though often not necessary; anemia, however, calls for the administration of iron. Return to ordinary avocations should be postponed as long as possible, especially after severe infections; return to mental work and study, in particular, should be delayed, since a too early return not infrequently results in a prolonged spell of cerebral asthenia. A sea voyage subserves a most useful purpose following convalescence.

Diet.—The diet should be suited to the individual patient, rather than to the disease. The physician should remember that during typhoid fever the digestive juices are lessened or perverted, and that some at least of the toxemia is due to the poisonous effects of the end-products of destructive katabolism of the body protein; and while he should endeavor as far as possible to supply a sufficiency of calories in foodstuffs to satisfy dynamic needs and to minimize loss of weight, he must be careful to avoid improper food and that excess of food which is certainly productive of digestive disorders. If in the past we have been perhaps too much committed to a diet insufficient in calories and relatively too rich in protein, a diet of too high caloric value may be productive of much harm; certain carbohydrates, under circumstances, may be of value; fats, at least in excess, are not well borne. In many respects the appetite is a good index of the amount, although not of the character of the food that may be permitted.

There can be no doubt that at present, as in the past, the majority of patients do best on a diet consisting largely, if not wholly, of milk. To an adult one may give four, six, or eight ounces of milk every two hours during the day, and every four hours at night—nine or ten feedings in the twenty-four hours. This furnishes from 780 to 1750 calories (650 to 700 calories to a quart or liter). The milk may be given hot or cold; it may be diluted with lime water or some carbonated water, such as Vichy; it may be flavored with salt, vanilla, coffee, tea, cocoa, or port or sherry wine; or it may be given as buttermilk, koumiss, matzoon, junket, or in part as ice cream. Twice a day, the white of an egg (20 calories) or the whole egg (80 calories), if well borne, may be added to the milk. In case of objection on the part of the patient, albumen water (the whites of two eggs added to four to six ounces of water, and flavored with lemon, lime, or orange juice) may be substituted for the milk at every other feeding. In ordinary cases, throughout the febrile period, I commonly adhere to this diet of milk and egg-albumen —the advantage of which is seen in the freedom from intestinal disorders, the good general condition of the patient, and the usually prompt convalescence without noteworthy untoward symptoms. However, in the event of very serious objection to milk on the part of the patient, or an obvious poor general condition, I have no hesitation in substituting the milk in whole or in part, or adding to it, cream, milk sugar, eggs, meat and

vegetable soups[1] strained and thickened with flour, powdered rice or barley, cream, egg, etc., or strained oatmeal gruel, cornstarch, arrow-root, potato puree, gelatine flavored with wine, etc.

In the event of tympanites or diarrhœa, the milk should be reduced in amount; usually, under these circumstances, too much rather than too little is being given, and curds frequently appear in the stools. Whey, boiled milk, peptonized or pancreatized milk, or koumiss may be substituted; but if the unfavorable symptoms do not subside promptly the milk should be entirely withheld for twenty-four to thirty-six hours, and albumen water given instead. Thereafter one may cautiously recommence with peptonized milk in small amounts, or resort to some of the other mentioned foodstuffs. If the patient has done well on the milk and egg-albumen dietary, toward the end of the third week, in the absence of definite indications to the contrary, two raw eggs daily should be added to the diet; and during the following week, the aforementioned gruels, broths, soups, cornstarch, etc., as well as very soft-boiled eggs. At the end of four to seven days of normal temperature, soft puddings, milk toast, scraped beef, mashed potato, apple sauce, etc., may be allowed; and a day or two later, sweetbreads, the white meat of chicken, stewed fruit, etc. The return to the ordinary diet should be gradual. Thus, while I incline to liberality in the matter of diet, especially toward the end of the disease, I have yet to see any general good come from the unusually generous dietary, even solid food, sometimes advocated, and I have seen it do positive harm.

Of quite as much importance as food is water. A minimum of three or four liters, in stated amounts between the feedings, should be given in the twenty-four hours; often much more can be taken with great advantage to the patient. The daily amount of urine is the best indication of the amount of water that should be given, and we may be sure that that patient who passes eighty, one hundred, or more ounces of urine daily will recover unless he suffers a severe hemorrhage or a perforation.

Hydrotherapy.—Hydrotherapy constitutes an essential part of the treatment of every typhoid-fever patient. Advocated originally by Currie, of England, hydrotherapy was systematized in 1861, and subsequently, by Brand, and is commonly known as the Brand treatment. Its advantages are many, the most important of which is not the reduction in temperature which it effects. Its greatest claim to our attention is that it unquestionably reduces the mortality—large statistics showing undoubtedly that from five to seven of every one hundred

[1] Houghton gives the following formula for the preparation of a vegetable soup: 60 grams (2 ounces) each of green or canned French peas, white dry beans, potato, rice, and noodles, and 15 grams (½ ounce) of carrot, should be boiled in water for at least four hours. Thereupon sufficient water should be added to make one liter (1 quart), which is sufficient for four feedings. This yields 760 calories, of which 6.3 per cent. is protein, 43.9 per cent. carbohydrate, and less than 0.2 per cent. fat. When ready to use, the sediment should be stirred up, and the patient allowed to eat everything except the pea- and bean-skins. Onion may be added for flavoring, if desired.

typhoid-fever patients are saved by its use. The patients saved are not the unfortunate subjects of complications such as hemorrhage and perforation, but patients who otherwise would die of toxemia. Hydrotherapy, therefore, does good by reducing toxemia, and largely through an influence on the nervous system.

Although, for obvious reasons, the degree of temperature is taken as the indication for repeating the hydrotherapeutic measures, the influence of the bath on the fever, especially during the fastigium, is often comparatively slight, though the toxemia may be notably influenced. Commonly, however, more or less bodily heat is abstracted; reduction of the temperature of from 2° to 4° after the baths is frequent, especially after the treatment has been in use for several days; and the ill effects of long-continued fever are obviated. The notable influence, however, is on the toxemia: the nervous manifestations of the toxemia, mental dulness, stupor, delirium, muscular twitchings, etc., are much improved, the "typhoid state" of our forefathers is rarely seen in patients subjected to the bath treatment from the beginning, and insomnia and coma vigil when present often after a bath give way to several hours of refreshing sleep; the toxic weakness of the heart and circulation (low blood pressure) is replaced by fuller, slower, and stronger heart beats, the blood pressure is increased, and peripheral cyanosis, hypostatic congestion of the lungs, venous thromboses, and other ill consequences of low blood pressure are warded off; shallow breathing gives way to full and deep inspirations, the cyanosis of ill-ventilation is lessened, the good results of improved oxygenation are obtained, and the bronchitis is improved, whence the likelihood of bronchopneumonia is lessened; the kidneys are stimulated to increased functional activity, and typhotoxins are excreted in increased amount; and the invigorating action of the baths and friction on the skin and the cutaneous capillaries improves the general tone of the system and diminishes the likelihood of bed sores.

Objections to the cold-bath treatment are: that it is troublesome —which is true, especially in private homes; that it is attended by no better results than other forms of treatment—which is not true; that it increases the likelihood of hemorrhage and perforation—which is not true, although certain series of cases may show somewhat higher percentage incidence of these complications: they show also a higher percentage of recoveries; that it is cruel, inhuman, and barbarous—which contains an apparent modicum of truth, but in reality the treatment does not add notably to, in many respects it reduces, the discomfort of the attack of typhoid fever, and assuredly it is better to suffer the ills of two or three weeks of cold-bath treatment than fly to those we know not of; and that it is not necessary in all cases—but the good results of the treatment are seen to best advantage only when it is instituted early and continued systematically throughout the attack, and not when postponed until the patient is "sick enough," a practice that unfortunately still obtains among some practitioners. No one can foretell at the beginning of an attack how severe it is likely to become.

The hydrotherapeutic measures to be commended in typhoid fever are the cold bath and sponging; the cold pack, sprinkling, and fanning are more or less unsatisfactory substitutes—available, however, in some cases.

The cold bath is the most preferable and is in general use in most modern hospitals, and as far as circumstances will permit is largely used by many physicians in their private practice. The common practice is to give the patient a bath at 70° F., for fifteen or twenty minutes, every three hours, if his temperature reaches or is above 102.5° F. While in the water, the patient's body should be submerged, a cold cloth or an ice-bag should be applied to his head, the exposed neck, shoulders, and chest should be douched repeatedly with cold water, and his extremities, back, and chest should be rubbed vigorously and continuously—an essential of the treatment. At the termination of the bath the patient should be wrapped in a dry, preferably warmed, sheet (previously, with a blanket, spread upon the bed); the sheet should be carefully tucked about the patient's extremities and trunk, and gentle friction made to remove the excess of water; hot-water bottles may be applied to his feet and legs, and then he should be covered with the blanket. After the patient is dry, the sheet may be removed and the patient allowed to rest in the blanket for twenty to thirty minutes, whereupon a dry nightdress should be provided. The rectal temperature should be taken at the termination of the bath, and again at the end of a half-hour. Usually a fall of temperature of from 1° to 2° will have been attained; this is sufficient, marked falls (4° and thereabouts) being undesirable.

Certain deviations from this strict regimen are not only permissible, but often desirable—due attention being paid to the patient rather than to the disease. Thus, it is by no means necessary that the water should be always at 70° F.; it may vary from 70° to 85° F., and it is often desirable to begin with a temperature of 85° F. in adults, and sometimes to continue with this temperature throughout the attack in children. Beginning with 85° in apprehensive adults, the temperature may be reduced 5° while the patient is in the bath, by the addition of ice or cold water—the cold water being preferable, since it is less likely to add to the apprehensiveness of the patient. Each succeeding or every second bath may begin at a temperature of 5° less, until the desired and the best temperature for the particular patient is obtained. Some patients, however, never do well with a temperature as low as 70° F. It is usually desirable to have the first bath last not more than five or ten minutes and gradually to lengthen the time of succeeding baths until the desired fifteen or twenty minutes is attained.

While in the bath some patients complain much of the low temperature; others shiver much, their teeth chatter, and they become more or less cyanosed; while others do not complain at all. Much depends upon the skill and tact with which the bath is given. Brand originally recommended that a glass of red wine be given the patient before and after

the bath; whiskey has been largely substituted in this country, but it is not necessary as a routine measure. In the event of unusual shivering or of cyanosis, especially of the face (rather than of the extremities, which occurs in practically all patients), a half-ounce (15 c.c.) of whiskey may be given; but equally good results attend the use of hot coffee, hot broth or milk, aromatic spirit of ammonia, Hoffmann's anodyne, etc. The patient should be removed immediately from the bath upon the occurrence of marked cyanosis (especially of the face), cardiac collapse, acute abdominal pain, vomiting, or intestinal hemorrhage. The baths are contraindicated in the event of severe abdominal pain, hemorrhage, perforation (peritonitis), cholecystitis, thrombophlebitis, unusual prostration, and cardiac weakness.

More than six tub baths are rarely required in the twenty-four hours. The patient should be allowed, if he will, to sleep at night; should the temperature be unusually high, cold sponging may be substituted for the bath at night. Following the suggestion of Dr. John H. Musser, I have been in the habit, for a number of years, of giving a tub bath for fifteen minutes when the temperature is above 103° F., and a sponge bath when the temperature is above 102° F.; thus while the treatments are increased in number, the number of tub baths, especially troublesome in private practice, are reduced. Various portable bath-tubs have been devised for use in private practice.

Cold sponging is an alternative hydrotherapeutic measure often necessary in private practice, and sometimes advisable in children and elderly persons. The water may be at 70° F., or less, even ice water, depending upon the indications in the case and the reaction of the patient. The general indications, and the frequency and duration of the spongings, are similar to those of the tub baths. While it is best to expose only that of the body being sponged, the patient should not be dried until the process is completed, since to insure reduction of the temperature a layer of water must be provided for evaporation.

The cold pack is sometimes serviceable in children. The patient should be wrapped in a sheet (previously doubled and wrung out of water at 60° to 70° F.), and then covered with a blanket—where he may remain for from thirty minutes to an hour. The patient sometimes sweats considerably, and the temperature may be reduced a degree or two; but the reaction and the influence on the toxic and nervous symptoms are, as a rule, not so marked as after the cold tub bath. Sprinkling the patient with cold water from a watering-pot, or by means of a spray-nozzle attached to an irrigating reservoir, is also efficacious in some cases. Evaporation may be facilitated by fanning the patient after the application of a wet sheet, which may be sprinkled from time to time. The application of continuous cold to the abdomen by means of an ice-bag or a Leiter's coil is favored by some practitioners, who believe that it reduces the temperature and lessens the likelihood of intestinal hemorrhage and tympanites. If none of the foregoing measures is resorted to systematically, the patient should be bathed at least once, prefer-

ably twice, daily, merely as a matter of cleanliness, to prevent bed sores, and to equalize the circulation.

MEDICINE.—Medicines should be given only when clearly indicated. I believe it good practice, though often not necessary, following ancient custom, if the patient comes under observation before the tenth day, and especially in the event of constipation, to give an aperient dose of blue mass (5 grains, 0.3 gram) or of calomel (5 to 10 grains at one dose, or $\frac{1}{8}$ grain, 0.008 gram, every fifteen minutes for eight doses), followed by an enema or a saline cathartic. In view of the common deficiency of gastric juice, and the occurrence of a dry, brown, coated tongue, it is advisable, especially in private practice, to give 10 to 15 minims (0.6 to 1 c.c.) of dilute hydrochloric acid, three times daily. Aside from this, many patients may require no medicine throughout the entire course of the disease.

The so-called intestinal antiseptics are of little use, since they do not achieve the object for which they are intended (the harmful bacteria are not those in the lumen of the intestine, but those in the lymphatic and the blood streams); they sometimes do some good, however, in preventing or limiting tympanites. The best is probably salol, which I believe I have seen do good. I have rarely seen any indication for administering carbolic acid, the mercurials, iodine, β-naphthol, menthol, thymol, guaiacol, chlorine, acetozone, etc. Those who desire only to reduce the temperature may use guaiacol, 30 minims (2 c.c.), painted on the skin of the abdomen and flank. Since I have never used any of the coal-tar antipyretics, except in an occasional dose, in the early stages of the disease, to relieve headache or severe neuromuscular pains, I am happy in the conviction that I have avoided doing my patients considerable, sometimes even fatal, harm—of which these drugs are unquestionably capable.

Alcohol should not be used as a routine measure, but should be withheld until it is clearly indicated, when without doubt it does considerable good. When the pulse becomes weak and dicrotic, the first sound of the heart weak and valvular; when insomnia is persistent and the fever high; and when the typhoid state develops, alcohol renders excellent service. It should be given carefully and not in too large amounts—beginning with a half ounce three or four times daily. In some cases the poor condition of the patient is due largely to the amount of alcohol given, and improvement immediately follows its discontinuance. Headache, in the early stages, is usually relieved by an ice-bag, sodium bromide, or a small dose of codeine or morphine; rarely one of the coal-tar derivatives, such as acetphenetidin or acetanilide, may be given in small doses, but a few doses only. Insomnia is best relieved by hydrotherapeutic measures, the patient not infrequently enjoying a refreshing sleep after the baths. General restlessness, particularly at night, is best controlled by a Dover's powder or a small dose of morphine hypodermicly. The bromides, codeine, chloralamide, trional, sulfonal, and similar remedies are sometimes useful.

Delirium and other manifestations of severe intoxication are best controlled by hydrotherapeutic measures. An ice-bag should be kept to the head continuously. Alcohol is useful, and is often required in relatively large amounts, but the caution already mentioned should be heeded. With the development of the typhoid state, stimulation must be more active, and special efforts must be made to rid the patient of toxins. Water should be given in very large amounts, and hot enteroclysis and hypodermoclysis (0.9 per cent. saline solution) are extremely serviceable. Cocaine, camphor, and codeine hypodermicly often control markedly the mental wandering, the carphologia, and the subsultus tendinum. Strychnine ($\frac{1}{40}$ to $\frac{1}{20}$ grain; 0.0015 to 0.003 gram) is often of value, but it should not be given continuously, since it often tends to aggravate the condition. In cases of marked cardiac asthenia with low blood pressure, the addition to the saline solution by hypodermoclysis of 2 to 4 c.c. of adrenalin chloride is often followed by good results. Digitalin germanicum (Merck), $\frac{1}{12}$ to $\frac{1}{6}$ grain (0.005 to 0.01 gram) hypodermicly, also often seems to do good, although it is said to lose much of its efficiency during states of fever. The meningism that much simulates meningitis is often markedly relieved by lumbar puncture.

Constipation rarely requires much treatment, and constipation as such is much to be preferred to diarrhœa. A simple soap-suds enema or an oil enema should be given every other day if the bowels do not move spontaneously. During the height of the disease no purgatives whatever should be given by the mouth. During convalescence special attention should be paid to the bowels, since, despite a daily evacuation, even of liquid feces, the patient may be constipated; that is, his rectum and colon may be more or less filled with hardened feces. The rectum, the urinary bladder, and the gall-bladder should be the special objects of concern in delayed convalescence. Well on in convalescence castor oil often subserves a useful purpose.

Diarrhœa should not be checked unceremoniously, not unless there are more than four movements in the twenty-four hours, and they are distressing and weakening to the patient. Since ill-digested food is usually the cause, the milk should be stopped, temporarily at least, and albumen water substituted. Bismuth, salol, and chalk mixture, or a lead and opium pill, or aromatic sulphuric acid and other astringents, may be administered for a few days, but care should be exercised that scybalous masses do not form. Tympanites, often associated with diarrhœa, is by no means so common as in the days prior to the cold-bath treatment. When developed it may usually be controlled by withholding the milk, temporarily at least, and administering turpentine externally (hot stupes), by the mouth, and by the rectum, if necessary. Charcoal, resorcin, β-naphthol, zinc sulphocarbolate, etc., by the mouth, and milk of asafœtida, by the rectum, are also sometimes of service. A rectal tube may be inserted high up in the sigmoid. A hypodermic injection of eserine ($\frac{1}{100}$ to $\frac{1}{50}$ grain, 0.0006 to 0.0013 gram) is sometimes efficacious

in causing the expulsion of considerable gas. Vomiting is rarely severe, and when present is usually controlled by withholding food for a time, and giving lime water with creosote or carbolic acid (in $\frac{1}{8}$ grain, 0.008 gram, doses), bismuth, cerium oxalate, sips of hot water, brandy, or dry champagne. Hot applications to the abdomen are also of service.

Hemorrhage is best treated by securing absolute rest to the patient and his intestines—by disturbing the patient as little as possible, even for evacuating his bowels, withholding all food for at least ten or twelve hours, and giving morphine hypodermicly. An ice-bag may be applied to the abdomen, except in the event of marked collapse and lowered temperature; custom sanctions the use of ice internally. How long the morphine should be continued must be determined in individual cases—remembering that, though it inhibits peristalsis, it favors intestinal distention, and in the event of perforation (associated in from 15 to 20 per cent. of the cases of hemorrhage) it masks the symptoms and may prevent their recognition. Turpentine is recommended to control repeated oozings. I have never seen undoubted good follow the use of any of the styptics, but lead (in the well-known lead and opium pill), Monsel's solution of iron sulphate, tannic acid, etc., may be used by those committed to the practice. Calcium chloride seems indicated theoretically to promote the coagulation of the blood, but its effects are lost after several days' use. Gelatine has largely gone out of use hypodermicly, but it may be given by the mouth, especially when food is recommenced, in the hope that it will favor the coagulation of the blood; but its action is at least doubtful. In the event of collapse, the foot of the bed should be elevated, hot-water bags should be applied about the patient, and hypodermoclysis and diffusible stimulants, such as ether and camphor, should be administered. Recently operation has been advised to control severe bleeding.

Perforation and peritonitis (which may be due to causes other than perforation) calls for immediate operation—now-a-days done largely under local anesthesia. The mortality without operation is at least 95 to 98 per cent., an occasional patient seeming to recover without operation, but in such cases the diagnosis must always remain more or less in doubt. With improved technique and early operation the mortality is becoming less and less, and is now probably not more than 60 to 65 per cent.; that is from 30 to 35 per cent. of patients are saved who otherwise would inevitably perish. Without operation, the treatment consists of supportive measures: opium to relieve pain, and hot water bottles, hypodermoclysis, and stimulants to combat shock and peritoneal sepsis.

Cholecystitis usually subsides without untoward symptoms, though it is usually the first of a train of serious symptoms (gallstones, etc.) that may develop in later life. If the symptoms are acute, however, if they increase rather than subside, and especially if there is reason to suspect suppuration or perforation, operation should be undertaken. In all cases the patient must be under continuous competent observa-

tion, so that should operation be called for it may be undertaken without delay. There is much to commend operation following recovery with a view to prevent the later development of gallstones.

Bed sores are better prevented than cured. Perfect cleanliness is imperative; the bed-linen should be well cared for and creases prevented; the patient should be turned frequently from side to side, and he should be bathed frequently with alcohol, alum water, etc.; and should any redness develop he should be placed upon an air cushion, and the reddened area painted with a 5 per cent. silver nitrate solution. If a bed sore develops, all pressure must be removed, by the use of the air-cushion, pads, or a water-bed or an air-bed; the ulcer should be treated antiseptically and with a drying dusting powder consisting of boric acid, zinc steorate, and bismuth subnitrate; all dead tissue should be removed, and if the granulations are sluggish, stimulating applications of silver nitrate should be made.

Thrombosis of the femoral or other veins should be treated by rest of the limb in an elevated position, and the application of cotton wool, an ichthyol ointment (33 per cent.), a lead and opium lotion, or a saturated solution of magnesium sulphate. In the early days an ice-bag over the thrombosis will relieve the pain. Later, mercury, belladonna, and iodine ointment (equal parts) is useful, but rough handling must be avoided on account of the possibility of embolism. Tender toes (peripheral neuritis) may be relieved by alcohol rubbings, massage, counterirritation (iodine, etc.), ichthyol ointment, and aconitine ointment (2 per cent.). Bacilluria, a frequent and unsuspected source of infection, is usually controlled by hexamethylenamine, 10 to 15 grains (0.6 to 1 gram) three times daily. It is advisable to give this as a routine measure for four or five days in the early days of convalescence. Should cystitis develop, we may use, in addition to the hexamethylenamine, irrigations of the bladder with mercuric bichloride, 1 to 50,000. As previously stated, the urine of every typhoid-fever patient should be disinfected.

PARATYPHOID FEVER.

Paratyphoid fever is an acute infectious disease caused by Bacillus paratyphosus (Archard and Bensaude); clinically it is characterized by symptoms that do not distinguish it from typhoid fever.

Etiology.—The term Bacillus paratyphosus is used as a convenient designation for a group of bacilli intermediate in biological type between the typhoid and the colon groups. Of the many subdivisions of these organisms, one much resembles Bacillus typhosus, another Bacillus coli, while others exhibit biological characteristics pertaining to Bacillus enteritidis, Gärtner (the meat poisoning bacillus), Bacillus suipestifer (hog cholera), and Bacillus psittacosis. Following Schottmüller and Buxton the paratyphoid bacilli are commonly divided into: (1) The paracolon bacilli, a group of organisms differing somewhat among

themselves but culturally very much alike, and, as a rule, not causing typhoid-fever symptoms in man; and (2) the paratyphoid bacilli, a group of organisms that do cause typhoid fever in man and are subdivided into Bacillus paratyphosus α, distinctly unlike the paracolon bacilli, and Bacillus paratyphosus β, resembling the paracolons, but a distinct species. Bacillus coli and Bacillus fæcalis alcaligenes also seem to be capable of causing a typhoid-like infection, so that the term typhoidal disorders is sometimes used to include this whole group of diseases. Aside from the specific cause, the etiological factors of paratyphoid fever are similar to those of typhoid fever.

Pathology.—The disease is a general bacteremia, and local lesions may be inconspicuous or entirely absent. The common noteworthy lesion is enlargement of the spleen. Otherwise the disorder resembles most of the bacteremias. The characteristic lesions of typhoid fever are usually absent; Peyer's patches and the solitary glands are slightly if at all altered; intestinal ulceration, in the few cases observed, has resembled dysenteric rather than typhoid ulceration; proliferative changes in the endothelium and endothelial phagocytosis, so characteristic of typhoid fever, have not been observed. Doubtless, many of the cases of so-called typhoid fever without intestinal lesions have been examples of paratyphoid infection.

Symptoms.—In general the symptoms are those of typhoid fever, but as a rule, paratyphoid fever has a shorter period of invasion, and the fever is less high, exhibits more marked diurnal variations, has a shorter course (twelve to twenty days), and ends rather rapidly (crisis or rapid lysis). Prolonged (eighty-four day), severe, and even fatal cases, however, have been observed. Diarrhœa appears to be somewhat more common than in typhoid fever. Intestinal hemorrhage occurs in from 5 to 10 per cent. of the cases. Pratt has emphasized the frequency of complications—which are often purulent in character. Relapses are about as frequent as in typhoid fever.

Diagnosis.—The disease is usually suspected to be typhoid fever until the Gruber-Widal reaction with the typhoid bacillus is found to be absent. The diagnosis depends then upon the recovery of the infecting microörganism—from the blood, the spleen, the urine, the feces, the sputum, the rose-spots, or the complicating lesions; or the diagnosis may be made by the agglutination reaction with known strands of paratyphoid bacilli.

Prognosis.—The mortality is low—not more than 3 per cent.

Treatment.—The treatment is similar to that of typhoid fever.

PYOGENIC INFECTIONS.

(*Pyococcic Infections; Septic Infections; Sapremia; Septicemia; Pyemia; Septicopyemia; Toxemia; Bacteremia.*)

The pyogenic infections comprise a group of non-specific infectious diseases, some local and some general, caused by the different pyogenic

microörganisms, and characterized by fever, leukocytosis, a variable degree of systemic intoxication, sometimes by bacteremia, chills, and sweats, and often by at least a local focus of inflammation or suppuration.

Etiology.—The pyogenic infections occur in all parts of the world and at all seasons; they are common at all ages, and under like conditions they affect the sexes equally—but for very obvious reasons women alone suffer from that form known as puerperal infection. While the robust are by means immune, these infections are especially common in persons debilitated from any cause, and they are often the terminal event in the course of many other diseases (terminal infections).

The exciting causes of these infections are especially the pyogenic cocci—Staphylococcus pyogenes aureus (citreus et albus), Streptococcus pyogenes, Diplococcus pneumoniæ, Micrococcus gonorrhœæ, Micrococcus intracellularis meningitidis, and Micrococcus tetragenus. Many bacilli and certain streptothrix also give rise to pyogenic infections, of which the more important are Bacillus coli communis, Bacillus typhosus, Bacillus pyocyaneus, Bacillus aërogenes capsulatus, Bacillus tuberculosis, Bacillus anthracis, Bacillus mallei, Bacillus pestis, and Friedländer's pneumobacillus. Although these bacilli unquestionably may produce suppuration, the pyococci are not infrequently associated with them, as well as with other bacilli (the diphtheria bacillus, for instance); that is, mixed infections are quite common. Infection may be transmitted by direct inoculation (such as infected sutures at operation), contact (such as necropsy and dissection wounds), or proximity (infected wounds and gangrene in hospitals and other infected localities), by a third person, and by fomites of all kinds. Susceptibility to the infection is much enhanced by a wound, abrasion, or traumatism of the skin or mucous membrane; in fact, were it not for the protection afforded by the skin and the mucous membranes these infections would be much more common than they are, since pathogenic staphylococci and often streptococci may readily be cultivated from the skin and certain mucous surfaces of the most human beings and lower animals, and they abound in the air, dust, and soil about us. Infection may be acquired by inhalation or ingestion, but it is much more commonly acquired by direct inoculation into the blood stream or the lymph stream (through a wound or abraded surface).

Pathology.—These pyogenic infections still labor under the embarrassment of descriptive terms born of ignorance and perpetuated in this day of comparative enlightenment—such as sapremia, septicemia, and pyemia. Sapremia was understood to be a condition due to poisoning by the chemical products of bacteria, but unattended by the entrance of the bacteria themselves into the blood; septicemia (septic infection, putrid material in the blood) was a condition due to the growth and development of microörganisms in the circulating blood, as well as to poisoning by their chemical products; and pyemia (pus in the blood), also called septicopyemia, comprised in addition to the foregoing the development of multiple abscesses in different parts of the body. And

when the source of the infection was not discoverable the condition was spoken of as cryptogenetic. These distinctions, however, are strained and artificial: except when introduced intentionally or taken in certain forms of food poisoning, poisoning of the body by the products of bacteria cannot occur independently of the presence of bacteria in the body, and contrary to former opinions, we now know that bacteria enter and circulate in the blood stream much more frequently than was formerly supposed, and that abrasions and other small and inconspicuous wounds of the skin and mucous membranes are frequently the infectious atria of what formerly would have been regarded as cryptogenetic sepsis or infection. Since, therefore, it is impossible, clinically as well as anatomically, to separate the one of these conditions from the others, since in reality they are but stages of the one process, it is much preferable to speak of them as pyococcic (or other) infections; when possible to qualify these by adjectives such as staphylococcic, streptococcic, pneumococcic, gonococcic, etc.; and to use the terms toxemia and bacteremia in their very obvious meanings. It is desirable to bear in mind also that it is usually impossible to distinguish between bacterial intoxication and bacteremia.

The pyogenic infections may be classified as follows: (1) Local infections; (2) general infections (*a*) with and (*b*) without obvious infectious atria; and (3) terminal or secondary infections.

Local Infections.—The local infections provoked by pyogenic microörganisms, especially staphylococci and streptococci, are legion, and comprise the inflammatory processes which arise in all parts of the body and which may or may not progress to suppuration, such as furuncle, carbuncle, abscess, phlegmon, cellulitis, erysipelas, lymphangitis, phlebitis, bronchitis, pleuritis, pericarditis, endocarditis, peritonitis, appendicitis, cholangitis, salpingitis, osteomyelitis, arthritis, puerperal infection, etc. As long as these (and other) infections remain local, the systemic intoxication is due solely to the soluble toxins produced by the bacteria and absorbed into the lymphatic and blood streams; in this respect these infections resemble certain specific infections, such as diphtheria and tetanus, in which the systemic symptoms are toxic solely. The severity of the symptoms, the toxemia, is proportionate to the amount and virulence of toxins absorbed, and since the toxin is being constantly excreted, especially by the kidneys, removal of the toxin-producing focus, such as a putrescent placenta, a pelvic abscess, an empyema, etc., results in almost immediate lessening of the toxic manifestations, and unless irreparable damage has already been done, recovery usually ensues. Contrary to former opinions, we now know that in many of these local infections there is a disposition to an early invasion of the lymphatic and blood streams, whence to the toxemia bacteremia is added. The bactericidal properties of the blood serum, however, are considerable, and though invasion of the blood may occur, general infection is often prevented.

General Infections.—Frequently on account of the virulence of the invading microörganisms the bactericidal properties of the blood are

overcome and general infection (bacteremia) occurs. This may or may not be associated with multiple abscess formation, whence it is spoken of as septicemia or pyemia, respectively. The term septicemia is also employed to designate the bacteremia that sometimes occurs in other infections, such as typhoid fever (typhoid septicemia), gonococcic infection, anthrax, etc.

These general infections may occur with or without an obvious local lesion. Much interest attaches to those cases, by no means rare, in which no local infection can be found during life or after death—so-called cryptogenetic infection or septicemia. Diligent search often results in the discovery of a small local lesion that otherwise would have gone undetected, but in some cases the most rigorous investigation fails to reveal any local source of infection. In the majority of cases, however, general infection is preceded by some local lesion, inconspicuous or apparently insignificant though it may apparently be, such as an acne pustule, a furuncle, a stitch abscess, a small abrasion; a latent or masked osteomyelitis, otitis media, adenitis, empyema, cholecystitis, or appendicitis; an unsuspected abortion, retained placenta, etc. Infection may be transmitted: (1) Along the lymphatics (which may or may not become actively inflamed); (2) by way of the venous circulation, in which event the venules frequently, and the larger veins sometimes, become thrombosed; and (3) sometimes by way of the arterial stream, especially in pulmonary or endocarditic lesions, or, as rarely happens, when a local focus of suppuration ruptures directly into an artery. Should the venous thrombi soften (a rather common event), the emboli carried to different parts of the body act either mechanically, producing infarction, or, if they contain virulent bacteria, setting up disseminated foci of suppuration: metastatic or embolic abscesses (so-called pyemia, or septicopyemia). Similar results ensue if the infectious matter is transported by the arterial stream. These metastatic phenomena occur especially: (1) In the lungs, particularly when the emboli have been transported by way of the systemic venous circulation; (2) in other parts of the body, when the arterial stream has become infected, either from rupture of a purulent focus into it, or from endocarditic thrombi, or from metastatic foci in the lungs, or by the passage of small emboli through the pulmonary capillaries; and (3) in the liver (suppurative pylephlebitis), when the primary focus is in the area of collection of the portal vein.

Terminal or Secondary Infections.—Terminal or secondary infections deserve special mention. In the majority of debilitating and wasting diseases, such as arteriosclerosis, chronic nephritis, cirrhosis of the liver, myocarditis, etc., the final event is determined not by the primary disease, but by some secondary or so-called terminal infection. This is usually caused by streptococci, staphylococci, or pneumococci, and less commonly by Bacillus coli, Bacillus tuberculosis, Bacillus pyocyaneus, Bacillus proteus, etc.; and the infection may be local or general. The local infections are the more common, especially pyococcic or tuber-

culous infection of the serous membranes—pleura, peritoneum, and pericardium.

In the milder pyogenic infections (pyococcic toxemia, sapremia) the lesions consist of—usually a local focus of suppuration, cloudy swelling of the organs, especially the liver, kidneys, and heart, enlargement and softening of the spleen, lessened coagulability of the blood, and often small hemorrhages in the serous membranes (pleura, peritoneum, and pericardium). In more severe cases, associated with bacteremia (septicemia), the foregoing lesions are aggravated: to the cloudy swelling of the organs foci of necrosis are added, the general lymphatics are enlarged and softened, and small hemorrhages appear not only in the serous membranes (including the pia-arachnoid), but also in the mucous membranes and the skin. In the cases usually described as pyemia, in addition to the foregoing, thromboses, especially in the region of the primary focus, and embolism with infarction and metastatic abscesses in different parts of the body, are found. Lesions of the endocardium (acute benign and malignant endocarditis) are common in all forms of pyogenic infections, and although they often materially modify the course and the symptomatology of the disease, they scarcely merit separate classification; in fact, the nature of the disease is better understood by bearing in mind that the involvement of the endocardium is a more or less accidental factor in the pyogenic infection, and that although the lesion may be serious the essential factor is the general pyogenic infection, of which the endocardial lesion may be the least important. The infective bacteria may be recovered (in pure or mixed culture) from the local lesions and the blood.

Symptoms.—The symptoms vary with the severity of the infection. Frequently the onset of the symptoms is insidious, and in case a primary local focus (furuncle, wound, puerperium, etc.) is present, a period of incubation, varying from one to three or four days, may be quite obvious. Occasionally the onset is sudden, with chill or chilliness, fever, malaise, headache, general neuromuscular pains and aching, and sometimes vomiting and diarrhœa. Especially characteristic is intermittent fever, with notable diurnal fluctuations (4° to 6° F.), accompanied by irregularly occurring chills and sweats (often the chief complaint). Sometimes the fever occurs in paroxysms of hyperpyrexia, between which the temperature may be of the continuous-fever type, or almost if not quite normal (apyrexia). In addition, the pulse is rapid and weak, the spleen is enlarged, diarrhœa may continue, and the headache gives way to restlessness, hebetude, stupor, and delirium; the patient becomes markedly prostrated, polynuclear leukocytosis is present, and a high-grade anemia develops rapidly. A subicteric hue or even jaundice is not uncommon, and certain cutaneous phenomena, such as roseola, erythema, urticaria, etc., doubtless toxic in nature, may be observed.

In the so-called pyemic cases, embolic phenomena (mechanical and septic infarctions) may occur in all parts of the body, especially: In the skin—petechial hemorrhages and furuncles; in the heart—ulcerative

endocarditis, septic myocarditis, and fibrinopurulent pericarditis; in the lungs—hemorrhagic infarcts (pain, dyspnœa, cyanosis, hemoptysis, localized dulness, bronchial breathing, and fine rales), abscesses, or fibrinopurulent pleuritis; in the kidneys—renal pain, albumin, blood, and tube casts in the urine; in the spleen—splenic pain, audible and palpable frictions; in the eye—retinal hemorrhages, choroiditis, optic neuritis, panophthalmitis; in the brain—hemorrhage (hemiplegia, monoplegia, aphasia, blindness, convulsions) or abscess; in the long bones—periostitis and osteomyelitis (local pain and tenderness); in the joints—arthritis (local pain, swelling, and tenderness).

The course of these infections is sometimes extremely rapid, especially when a large amount of toxin is suddenly thrown into the circulation; death may result within twenty-four or forty-eight hours. Commonly the symptoms last for from one to three weeks; occasionally several months.

Diagnosis.—A primary focus of infection, irregularly occurring fever, chills, and sweats, prostration, leukocytosis, anemia, cutaneous hemorrhages and other embolic phenomena, render the diagnosis easy in the majority of cases. Confirmation may be obtained by cultivating the infective microörganisms from the blood. Involvement of the endocardium (malignant endocarditis) is suggested by embolic phenomena and instability of the cardiac activity (rapid changes in frequency of beat). Malaria may be excluded by the presence of leukocytosis, the irregularity of the rigors and sweats, absence of the plasmodium, and the non-response to quinine. In acute tuberculosis, as contrasted with pyogenic infection, variations in the temperature and pulse-rate and excessive perspirations occur with more regularity, wasting is more rapid, a local focus of tuberculous disease may be detected in some part of the body—in the lungs (tubercle bacilli in the sputum), in the pleura, in the abdomen, in the brain (tubercle bacilli in the cerebrospinal fluid obtained by lumbar puncture)—and embolism does not occur. The detecting of chloroidal tubercles in the one case, and of retinal hemorrhages in the other, is of the greatest diagnostic value. In case of doubt as regards typhoid fever, persistent headache, persistent but not intermittent fever, comparative infrequency of the pulse, roseola, tympanites, the Gruber-Widal reaction, the recovery of typhoid bacilli from the urine, feces, roseola, blood, etc., and the absence of petechiæ and of embolism, suggest typhoid fever, whereas pyogenic infection is suggested by a local source of infection, undue dyspnœa, irregular and repeated rigors, profuse sweats, early marked prostration, the rapid development of anemia, leukocytosis, embolism, and the recovery from the blood of pyogenic microörganisms. In obscure cases of evident intoxication, hidden or latent foci of infection, such as gonorrhœa, prostatic abscess, pyelitis, osteomyelitis, cholecystitis, etc., should be diligently searched for. We are not yet able to distinguish clinically between the different pyogenic infections, but in general the streptococcic infections are the more serious,

give rise to a more rapid and progressive anemia, greater prostration, and often an unusual mental alertness.

Prognosis.—The outlook depends upon the severity of the infection, the resistance of the patient, and the possibility of removing the infectious foci. Remarkable improvement often ensues immediately after the local foci are removed; if, however, the patient's tissues are already seriously damaged, this may avail little, if anything.

Treatment.—The prime indication is to remove the infectious foci—whence the treatment is largely surgical. Little can be expected of medicinal measures if the remnants of a putrid placenta are allowed to remain in the uterus, or if a liver abscess, brain abscess, or an empyema is permitted to go undrained. In addition to surgical measures, the patient's strength must be conserved, by fresh air, sunshine, and good nutritious food. Whiskey, quinine, iron (tincture of the chloride), and strychnine are useful adjuvants—supportive and restorative, and hot saline solutions (hypodermoclysis and enteroclysis) assist in eliminating the toxins. The coal-tar antipyretics should never be employed. The sweats may be controlled by alcohol baths, atropine, and aromatic sulphuric acid. Credé's colloid silver (1 per cent. solution, hypodermically and intravenously) has been suggested, but the results have not been very encouraging. Antistreptococcic serum has not proved of great service. The best results recently have followed the use of autogenous vaccines (bacterins); that is, isolating from a local lesion or the patient's blood the infecting microörganism and from this preparing a vaccine.

ERYSIPELAS.

(*St. Anthony's Fire.*)

Erysipelas is an acute, perhaps specific, infectious, and moderately contagious disease caused by Streptococcus erysipelatus (Fehleisen), and characterized by high fever, marked toxemia, and inflammation of the skin and subcutaneous and the mucous and submucous tissues. The disease in reality is merely one manifestation of pyococcic infection.

Etiology.—Erysipelas occurs at all ages, being common in early life, in adolescence, and in adult life; but it is rather uncommon in old age. It affects males more frequently than females. It is endemic in most large cities, and frequently prevails epidemically, especially during the late winter and spring; but sporadic cases are common. Recurrences are rather frequent; indeed, in some persons one attack seems to predispose to subsequent attacks.

The exciting cause of the disease is Streptococcus erysipelatus, which, however, is now believed to be identical with Streptococcus pyogenes. Infection may be transmitted by direct inoculation, contact, or proximity (possibly transmitted by the air), by a third person, and by fomites—whence the frequent occurrence of epidemics (less common recently than in former years) in insanitary hospitals, institutions, barracks, etc.

Susceptibility to the disease is much enhanced by a wound of the skin or of a mucous membrane—whence the frequent occurrence of the disease (traumatic erysipelas) after parturition, operations, traumatic wounds, etc. In many of the so-called idiopathic cases a history of previous nasal catarrh may be elicited, and in many others there are doubtless unsuspected wounds or abrasions of the nasal or oral mucous membrane—whence the frequent occurrence of facial erysipelas. Debilitating influences, such as those connected with chronic alcoholism, chronic nephritis, cirrhosis of the liver, etc., also favor the development of the disease. Infection apparently is usually acquired directly into the blood or the lymph channels.

Pathology.—The lesions consist of the ordinary changes of inflammation with marked œdema and cellular infiltration. The streptococci are found especially in the lymphatic channels (lymphangitis), and in large numbers in the zone of spreading inflammation and beyond (where active phagocytosis may be observed). The visceral lesions are those common to severe pyococcic infections. Suppuration of the serous membranes is rather common—pleuritis, pericarditis, endocarditis, meningitis, arthritis, etc.

Symptoms.—In so-called idiopathic (non-surgical) erysipelas, the period of incubation varies from three to seven or ten days. Often prodromal symptoms are absent, but usually there is headache, malaise, restlessness, general aching, and loss of appetite; possibly also slight elevation of the temperature. The onset is usually abrupt with a chill or chilliness (which may be repeated), high fever (104° to 105° F.), sometimes nausea and vomiting, followed soon by the specific cutaneous manifestations. These develop first on the bridge of the nose or on one cheek near the nose, or at a mucocutaneous junction (in the event of a wound, however, immediately about the wound). They consist at first of slight redness; at the end of twenty-four hours, however, the skin has become red and hot, smooth and glistening, swollen and quite tense (inflammatory œdema). The lesions spread by peripheral extension, and are usually sharply demarcated from the adjacent unaffected skin by a well-defined, elevated, rather pale, and indurated ridge. Often the cheeks, the eyelids, the forehead, the ears, sometimes the entire scalp and even the neck become involved, and give rise to extreme disfiguration. As the disease spreads, the swelling and redness of the parts first affected gradually subside. Blebs not infrequently form, especially on the eyelids, forehead, and ears. The cervical lymph nodes are generally much enlarged.

The systemic disturbance varies much in different cases; it is often remarkably slight, as contrasted with the rather severe cutaneous manifestations, but it may be extreme. Usually the fever remains high, with slight remissions, for four or five days, and then falls by a rather rapid lysis (sometimes even a crisis). The pulse is rapid; albumin is frequently present in the urine; and there is often stupor and a moderate amount of delirium. Leukocytosis is present. In favorable cases the subsidence of the cutaneous lesions is followed by desquamation (the scales being

infectious), and the patient enters upon convalescence. In unfavorable cases, the fever remains high, the typhoid state develops, the delirium becomes more marked, the urine lessened in amount, the albuminuria more marked, and in convulsions or coma the patient may die. In severe cases that do not go on immediately to death, more or less extensive suppurative (phlegmonous) inflammation, and in some cases, even gangrene, supervenes. Small abscesses are quite common in many otherwise mild cases.

An unusual form of erysipelas has been described—in which the cutaneous lesions spread from the face to the neck, to the chest, and may even gradually involve the entire body—erysipelas migrans. Occasionally the lesions involve the mucous membrane of the nose and mouth (usually secondarily, but sometimes primarily), and occasion an erysipelatous faucitis which may go on to the most extensive phlegmonous inflammation and lead to œdema of the glottis, otitis media, inflammation of the eye and the other orbital tissues, etc. More or less swelling and redness of the fauces, however, is not uncommon in cases of ordinary erysipelas, and œdema of the glottis may result from extension of inflammation from without (from the neck).

Complications.—Pneumonia, pleuritis, pericarditis, malignant endocarditis, meningitis (rarely, however, from extension through the skull, as erroneously believed), arthritis, nephritis, and metastatic abscesses are the more common complications.

Diagnosis.—As a rule, the disease is readily recognized. Erythema, urticaria, and acute eczema should be distinguished. Erysipeloid of Rosenbach (erythema migrans) occurs especially in those who handle meat, crabs, and other shellfish, and occasionally in laboratory workers; it is characterized by an area of inflammation, usually on the hands, sharply outlined, dark, reddish purple in color; and it usually subsides within ten days or two weeks.

Prognosis.—The disease lasts usually ten to fourteen days. The mortality varies from 4 to 7 per cent. The prognosis is bad in newborn infants, in whom the region about the umbilicus is often involved, in unusually severe infections, and in aged, debilitated, and alcoholic subjects. Recurrences which are not uncommon are frequently due to unsuspected erosions of the nasal mucous membrane.

Treatment.—The regulations enforced in infectious diseases (page 29) should be carried out. The patient should be confined to bed, and given a light, nutritious diet. Since the disease usually pursues a self-limited course, it is doubtful if medicines exert any specific influence; but I am in the habit of giving quinine and iron, which are sanctioned by age and long experience, and seem to do good:

℞—Quinine sulphate	48 grains	3	0
Tincture of iron chloride	6 drams	22	5
Glycerin	4 drams	15	0
Distilled water, sufficient to make	6 ounces	200	0—M.

S.—Two teaspoonfuls (10 c.c.), in water, four times a day.

In mild cases stimulants are not required, but alcohol and strychnine should always be given in severe infections, in the aged, and in the debilitated. Da Costa obtained good results with pilocarpine subcutaneously, $\frac{1}{6}$ grain (0.01 gram) every two or three hours for three or four doses, in the early stages; but assuredly it should be given only to sthenic subjects, and never when the disease is advanced. The coal-tar products are absolutely contra-indicated. Antistreptococcic serum has not yet sustained the claims originally made for it. Better results are to be expected from the use of autogenous vaccines (bacterins).

Locally a saturated aqueous solution of magnesium sulphate, applied on a gauze mask, and renewed twice in the twenty-four hours, is the best form of treatment; but one may use also applications of cold water, lead water and laudanum, mercuric bichloride (1 to 4000), carbolized vaseline, or ichthyol ointment (33 per cent.). The injection of carbolic acid (2 per cent.), mercuric bichloride, or mercuric biniodide into the area of advancing œdema has been practised with some good results and many failures; this is also true of painting the area with silver nitrate. Judd extols the value of painting the inflamed area and one-half inch of the surrounding apparently healthy skin with 95 per cent. carbolic acid, and after the painted region has become white applying pure alcohol until it becomes pink. Should suppuration ensue, the pus should be evacuated surgically.

For the nervousness, headache, and insomnia, one may use the bromides, chloral, opium, and hyoscine. Large amounts of water should always be given (as in every toxic process) to facilitate elimination of the toxins. The fever, if high, calls for hydropathic measures —upon general principles.

PNEUMOCOCCIC INFECTIONS.

By pneumococcic infection is understood an infection of the body by the pneumococcus, a microörganism first discovered by Sternberg, in 1880, later studied by Fraenkel and Weichselbaum, and sometimes described as Diplococcus pneumoniæ, or Micrococcus lanceolatus. The most common manifestation of such infection is the ordinary croupous or lobar pneumonia, of genuine cases of which the pneumococcus is probably the sole etiological factor. The pneumococcus, however, is causative of many other diseases, such as bronchopneumonia, pleuritis, pericarditis, endocarditis, peritonitis, meningitis, arthritis, otitis, etc. Although these are sometimes primary, they are commonly secondary infections, and they may be the result of mixed infections. A number of terminal infections in other diseases also are due to the pneumococcus. Furthermore, a distinct pneumococcic infection without local lesions, a pneumococcic septicemia (bacteremia), may occur. This, which is somewhat comparable to typhoid septicemia, typhoid fever without intestinal lesions, is usually sudden in onset with chill and fever, but

with few if any local symptoms, and it progresses rapidly to a fatal termination under manifestations of profound intoxication—coma and collapse of the circulation. The pneumococcus may be cultivated from the blood and other tissues of the body.

PNEUMOCOCCIC INFECTION OF THE LUNG; PNEUMONIA.

(*Pneumonitis; Croupous, Fibrinous, or Lobar Pneumonia; Lung Fever.*)

Pneumonia is an acute, specific, infectious, and mildly contagious disease caused by Diplococcus pneumoniæ (Sternberg, Fraenkel-Weichselbaum); it is characterized anatomically by bacteremia and a local inflammation in the lung proceeding to solidification, and clinically by initial chill, rusty expectoration, and high fever that usually ends by crisis.

Etiology.—Pneumonia occurs at all ages, being common prior to the sixth year and in adult life and old age. It is less frequent during adolescence than at any other period of life. It affects males more than females, and the urban more than the rural population; and, while it frequently attacks those apparently in robust health, it is especially prone to attack those debilitated by fatigue, starvation, unhygienic surroundings, exposure to the inclemencies of the weather, and overindulgence in alcohol, and those subject to or convalescent from certain diseases, such as, influenza, typhoid fever, Bright's disease, diabetes, etc. It sometimes follows traumatism to the chest (traumatic pneumonia), and it frequently follows "catching cold"—although this is now thought to act merely by reducing the resistance of the individual. Recurrences are common, one attack seemingly predisposing to others. It is especially fatal in the negro.

Pneumonia prevails in all parts of the world, and may be said to be endemic in most large cities. It is especially prevalent during the winter and spring, more particularly in February and March, when marked and sudden variations in temperature and humidity are common. Small epidemics, especially house (jail, barrack, ship, etc.) epidemics are not infrequent.

The exciting cause of the disease is the pneumococcus, which may be found in the lesions in the lung, in the secretions of the entire respiratory tract (sputum), in the blood, and sometimes in the complicating lesions. It has been found also in the mouths of healthy persons, as well as in the dust and hangings of rooms. Pneumonia is slightly contagious—some outbreaks more so than others. Infection is usually transmitted directly, by a third person, or by the air, although it may be transmitted by fomites; it is usually acquired by inhalation, the lung being first affected. There is some evidence, however, that the blood may be primarily infected, through a wound or the tonsil, for instance, and the lung, or other organ, secondarily. Secondary infections with the ordinary pyogenic microörganism are not uncommon.

The pneumococcic process may also be set up by other microörganisms, such as Friedländer's pneumobacillus, the influenza bacillus, the typhoid bacillus, the plague bacillus, streptococci, staphylococci, etc., but the processes induced by these microörganisms differ somewhat anatomically, as well as clinically, from true pneumococcic infection of the lung.

Pathology.—The pneumonic process involves the right lung more frequently than the left, because the right bronchus is larger and more in a straight line with the trachea than the left, and the lower lobe more frequently than the upper. Involvement of the upper lobe in adults is often more serious and more likely to terminate fatally than involvement of the lower lobe; but the upper lobe is often involved in children and usually ends favorably. In some cases both lungs are involved, whereas in other cases first one portion and then another portion of the lung becomes involved.

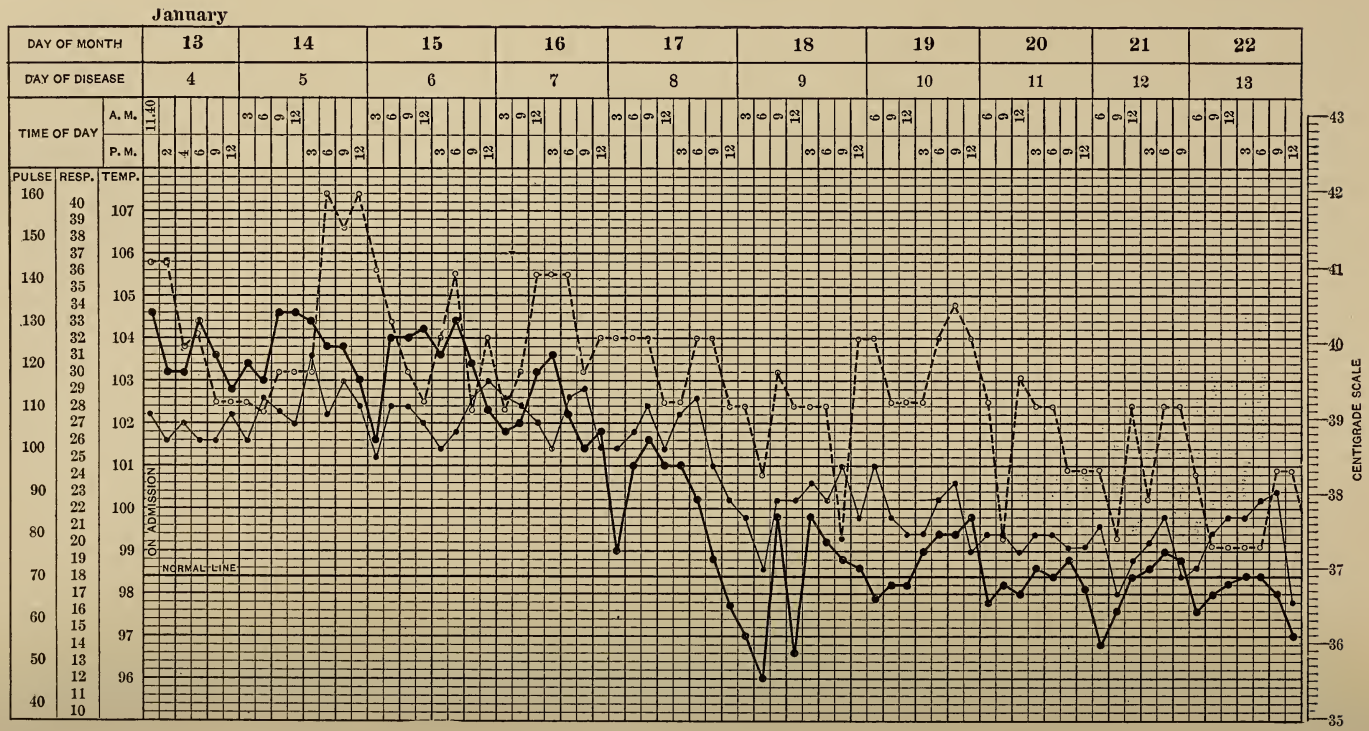
Four stages of the process may be described: (1) Congestion; (2) red hepatization; (3) gray hepatization; and (4) resolution. Frequently, however, the lung reveals several stages side by side. In the first stage, or stage of congestion, the lung is markedly congested, dark red in color, and of increased consistence, although it still crepitates and floats on water. The capillary bloodvessels are much congested and dilated—protruding into the alveolar air spaces, which are much encroached upon, and which, together with the infundibula and the commencements of the bronchi, are more or less filled with a tenacious exudate containing erythrocytes, a few leukocytes, and desquamated alveolar epithelium. As the disease progresses, the exudation increases until it entirely displaces the air in the alveoli—the second stage or stage of red hepatization. Not only the fluid, but also the cellular exudate, consisting of erythrocytes, mononuclear and polynuclear leukocytes, desquamated epithelium, and pneumococci, is increased in amount, and the entire exudate becomes coagulated to form a fibrinous mass—whence the term fibrinous or croupous pneumonia. The interstitial tissues of the lung also show inflammatory phenomena. In this stage the affected portion of the lung is increased in consistency, weight, and volume (often revealing indentations from the ribs); it is dark, reddish brown in color (although less congested than in the first stage); it no longer crepitates, and it sinks in water—in a word, it much resembles liver tissue, whence the term hepatization. The section surface is congested (mottled), dry, dull, granular (due to the fibrinous plugs in the alveoli), and the organ is quite friable. Red hepatization gradually merges into gray hepatization—evidence of increased leukocytic infiltration, lessening of the congestion from pressure upon the bloodvessels by the exudation, and beginning autolysis of the exudate. The lung becomes mottled, grayish red in color, its surface less granular and more moist than previously—the result of degeneration and necrosis of the exudate and of the transudation of serum from the bloodvessels. The exudate consists in large part of polynuclear leukocytes (phagocytes), although degenerated erythrocytes and desquamated epithelium

may be distinguishable. As the exudate becomes softened, in consequence of autolytic processes, it is removed, in large part by absorption, in small part by expectoration; resolution occurs, and gradually the lung returns to its normal condition. In less favorable cases the lung may become the seat of purulent infiltration—diffusely infiltrated with polynuclear leukocytes and appearing yellowish, soft, and puriform to the naked eye. In other cases the lung process goes on to abscess formation (one large abscess or several small abscesses that usually fuse to form a large one)—due often to secondary infection with streptococci and other pyogenic microörganisms; or a portion, or an entire lobe, becomesg angrenous—due to defective blood supply and to secondary infection with putrefactive bacteria. In other cases resolution is delayed and may be followed by induration of the lung or tuberculosis.

The non-solidified portion of the lung may be normal, but it usually shows more or less congestion and œdema. Sometimes there is an associated bronchitis, and the fibrinous plugs may extend far into the bronchi, forming veritable casts. The pleura investing the affected portion of the lung is almost always more or less inflamed, revealing usually a fibrinous exudate, although the lesions may progress to serous-fibrinous exudation (pleuropneumonia; metapneumonic pleuritis), or to suppuration (metapneumonic empyema). The organs in general show the results of toxemia and infection. Thus, the bronchial lymph nodes are swollen and soft, the myocardium, kidneys, and liver the seat of parenchymatous and sometimes fatty degeneration, and the spleen is swollen. The heart is frequently markedly dilated (especially the right side), and it may contain firm coagula (attributable to increased fibrin in the blood), which may lead to sudden death. Complications such as pericarditis, endocarditis, meningitis, enteritis, and other evidences of infection are not infrequent.

Symptoms.—The period of incubation is not definitely known, although it probably varies from a few hours to several days. Usually there are no prodromal symptoms, but in some cases the patient may complain for a day or two of slight catarrh of the upper respiratory tract, malaise, loss of appetite, headache, and dull pains in the limbs. In the majority of the cases, however, the onset of the disease is sudden—usually with a severe chill, but sometimes in children, with a convulsion or vomiting. Concurrent with the chill the temperature rises rapidly and attains its maximum, 104° F. or more, within a few hours. At the same time the patient complains of headache and general muscular or neuromuscular pains, severe stabbing pain in the side, cough, rusty expectoration, dyspnœa, and the usual concomitants of fever, such as rapid pulse, dry skin, a flushed face, loss of appetite, concentration of the urine, etc. Soon the physical signs of solidification of the lung and the general evidences of toxemia with delirium become apparent. The clinical manifestations continue usually unabated for from five to nine days, in the majority of cases, when the crisis occurs, and the patient enters upon what is usually a rapid convalescence.

FIG. 2



The temperature (heavy continuous line), the pulse (light continuous line), and the respiration (broken line) in pneumonia. (Personal observation, University Hospital, Professor Musser's service.)

THE FEVER.—The fever of pneumococcic infection of the lung is quite characteristic (Fig. 2). As already stated, it rises rapidly and may reach 104° F. or more, within a few hours. In most, especially asthenic, cases the temperature is usually stable, that is, for several days the diurnal variations may be not more than a degree. By the third day, however, there may be a slight remission and the subsequent diurnal variations may amount to 1.5° F., but the temperature remains high until some time between the fifth and ninth days (usually the fifth, seventh, or ninth, rarely the third, day) when the characteristic crisis occurs—a sudden fall of the temperature, within two to ten hours, to normal or less, accompanied by profuse sweating, lessening of the dyspnœa and the general toxic manifestations, increase in the strength of the pulse and the amount of urine, and marked improvement in the general condition of the patient, without however, in many cases, any noteworthy changes in the physical condition of the lung. Frequently the crisis is followed by a prolonged and refreshing sleep. Occasionally the crisis is unduly protracted—beyond twenty-four hours. Not infrequently on the day or the second day preceding the crisis the temperature falls to between 99° and 101.5° F. and rises again—a pseudocrisis. On the other hand, in cases with prodromal symptoms and sometimes in children the onset may be gradual, and in debilitated and alcoholic subjects the temperature range is likely to be relatively low—102° to 102.5° F. In cases protracted beyond the tenth day the fall of temperature is frequently by lysis. In some cases the temperature rises before the crisis—a so-called precritical rise. Such rise, however, may be merely a forerunner of death, but in most cases the temperature falls just before death. Aside from the precritical hyperpyrexia, the temperature may be unusually high throughout the course of the disease—106° to 107° F. (usually fatal cases); in other cases there is no elevation of the temperature whatever—afebrile pneumonia.

RESPIRATORY SYMPTOMS.—Dyspnœa is a constant and characteristic symptom, the respirations varying from 30 or more per minute on the first day, to 50 or 60 per minute on the third or fourth day; sometimes however, reaching 80 to 100 per minute in children. The breathing is superficial, repressed, and evidently painful; and the expiration is usually accompanied by an almost pathognomonic grunt. The dyspnœa is not entirely dependent upon the solidification of the lung, being due also to pain, fever, toxemia, and loss of functionating lung surface; it is frequently out of proportion to the extent of the lung involvement and it disappears with the occurrence of the crisis, although the physical condition of the lung may persist unchanged for several days. The pulse-respiration ratio may become 2 to 1, even 1.5 to 1, instead of the normal 4 to 1. Pain is an early and distressing sign, and is usually attributable to associated involvement of the pleura. It is severe, stabbing in character, and much increased by deep inspiration and by coughing. It is generally referred to the region of the nipple or the lower ribs on the affected side, rarely to the opposite side, or to the abdomen, back, or

shoulder. It gradually disappears after the third or fourth day. It may be slight or absent in central and in apical pneumonias. Cough and expectoration are initial symptoms. In the early stages the cough is short, harsh, and restrained on account of the pain that it provokes. The expectoration, which may be mucous in character for a few hours, speedily takes on almost pathognomonic characteristics, becoming rusty, viscid, and tenacious. Rarely there is a distinct hemoptysis. In certain asthenic cases, the sputum may be thin, less viscid, and darker (more hemorrhagic) in color (prune-juice sputum). After the crisis the cough usually becomes less harsh and the sputum less viscid and more mucopurulent in character. Cough and expectoration may be almost, if not entirely, absent in certain asthenic cases, in much debilitated and alcoholic subjects, and in the very young and the very aged. Microscopic examination of the sputum reveals Diplococcus pneumoniæ (often in pure culture, though frequently associated with other microörganisms), erythrocytes, leukocytes, mucus, alveolar epithelium, and sometimes fibrinous plugs from the alveoli and casts of the finer bronchioles.

Circulatory Symptoms.—In ordinary cases the pulse is usually 100 to 110 or 120, in children 140 to 160, per minute, and full and bounding in the height of the disease. Late in the disease, especially if the attack be severe, the pulse loses its strength and becomes more rapid (160 or more per minute). A pulse of more than 120 per minute, as well as a small, weak, and irregular pulse, always occasions concern. In ordinary cases the heart sounds are clear and the pulmonary second sound accentuated. In the height of the disease functional murmurs are common; with dilatation of the heart due to weakness of the myocardium, the result of toxic degeneration and excessive pressure in the pulmonary circulation, relative insufficiency of the mitral and tricuspid valves may develop. Early evidence of cardiac embarrassment is furnished by lack of muscular tone of the first sound of the heart and weakening of the previously accentuated pulmonary second sound; later, there may be an extension of the cardiac dulness to the right, epigastric pulsation, and embryocardia. Sudden collapse of the circulation—weakness and irregularity of the heart and the pulse, low blood pressure, subnormal temperature, cyanosis, and sweating, are by no means unknown, and are the result of toxic paralysis of the vasomotor centres, toxic degeneration of the myocardium, and excessive pressure in the pulmonary circulation. Though it may prove fatal, it may be recovered from.

Blood.—Polynuclear leukocytosis is a diagnostic feature of most cases. The leukocytes usually number from 18,000 to 45,000 per c.mm.; they may be more numerous (rare), and in extremely asthenic and toxic cases there may be little or no leukocytosis (a bad prognostic sign). Leukocytosis begins with the onset of the disease, persists throughout its course, and falls with and after the true crisis. During the height of the disease the eosinophiles are much reduced or absent; they reappear with or just prior to the crisis. The other blood changes consist of a secondary oligocythemia and oligochromemia, rapid clotting

of the blood, due to increase of the fibrin elements, and the presence of the diplococcus in some cases.

Nervous Symptoms.—Nervous symptoms are a characteristic of all cases. Headache, mental dulness, and at least slight mental wandering, are rarely missed. In well-developed cases, delirium is constant, although it varies in character. It may be maniacal, but it is usually of a low adynamic muttering character (profound toxemia), or it may resemble delirium tremens (true delirium tremens is the rule in alcoholic subjects). Convulsions are common at the onset in children, and some cases of pneumonia in children run their course altogether under the guise of meningitis (so-called cerebral pneumonia). Cerebral symptoms sometimes develop about the time of, or persist after, the crisis, and, consisting of mental hebetude, delirium, rarely of insanity, they may continue for several days.

Digestive Symptoms.—Initial vomiting is common, especially in children. The tongue is coated with a white or yellowish white fur and later sordes not infrequently collect upon the teeth and gums, and the tongue may become fissured. Thirst is marked, the appetite is lost, and the patient is usually constipated, unless enteritis or colitis develops. Meteorism, sometimes of dangerous grade, since it embarrasses the already embarrassed heart and lungs, may accompany the pneumococcic enteritis or colitis, or it may be due to toxic paresis of the intestine. The spleen is usually enlarged and palpable, and the liver can often be felt, especially in right-sided pneumonias, in which an apparent enlargement may be due only to displacement of the liver downward.

Cutaneous Symptoms.—Herpes occurs in about one-third of the cases on the lips and about the alæ of the nose, but occasionally also on the cheek, ear, arms, genitals, etc. It develops usually on the second, third, or fourth day of the disease, and sometimes comes out in successive crops. Moderate sweating is common throughout the latter half of the disease, and profuse sweating occurs with the crisis. A dusky flush on the cheek, said to be homolateral with that of the lung affected, is quite common; sometimes the flush occurs on both cheeks, being more marked on that of the side affected with pneumonia.

Urinary Symptoms.—The urine presents the characteristics of febrile urine—concentrated, lessened in amount, high colored, increased in specific gravity, and depositing a copious sediment of urates and uric acid. Albumin, referable to toxic changes in the renal epithelium, is common; sometimes a true nephritis (much albumin, blood, and tube casts) develops. The chlorides are usually much diminished, or absent throughout the height of the disease. The diazo reaction is sometimes present.

Physical Signs.—In the first stage, or stage of congestion (usually of not more than twenty-four hours' duration), the respiratory movements over the affected portion of the lung are restricted; there is slight increase of tactile fremitus and of vocal resonance; the percussion note may be normal, but is usually higher pitched and somewhat tympanitic

(Skodaic resonance); and the breath sounds are weak or bronchovesicular in character. The crepitant rale of Laënnec may be heard. This is usually said to be due to forcible separation of the alveolar walls and infundibula rendered adherent by the viscid exudation; in some cases, however, a sound indistinguishable therefrom is more likely a pleural friction, since it is not heard in all cases, and not until the end of the first stage, when the pleura may be covered with a layer of fibrin.

In the second stage, or stage of solidification, the respiratory movements over the affected portion of the lung are much restricted, while those over the other lung are exaggerated; the affected side may be somewhat enlarged (1 to 1.5 cm.), but the intercostal depressions are not obliterated. Tactile fremitus and vocal resonance are much increased, except in the exceptional cases in which the bronchi are plugged by mucus or fibrinous exudate, or when there is an associated pleuritis. The percussion note is dull—there being varying grades of dulness from the slight dulness of the first stage to marked dulness or flatness, present especially posteriorly, in the fully developed disease. With the dulness there is increased resistance to the percussing finger, but neither the dulness nor the resistance are as marked as in pleuritis with effusion. Dulness is less marked in incomplete solidification, especially in pneumonias that do not fully reach to the surface (central pneumonias), in the very young, and in the aged (on account of senile changes in the ribs and cartilages). Skodaic resonance may be elicited above the solidified area. The breath sounds are bronchial or tubular in character, though rarely they may be absent in case the bronchi are plugged with exudate; a voluntary cough, however, will usually dislodge such plug, which, being followed immediately by bronchial breathing, is conclusive evidence of solidification. Bronchophony, sometimes egophony, may be elicited (unless the bronchi be plugged). I have known Wintrich's change of note and amphoric breathing to lead to confusion with a cavity. In associated bronchitis, fine, crackling rales may be heard, and usually with undue distinctness on account of the solidification. The uninvolved lung reveals exaggerated vesicular breathing, and occasionally, especially in children and young subjects, bronchial breathing due to conduction from the diseased lung.

With the onset of resolution there is a gradual return to the normal conditions. Movement returns to the previously immobile chest, tactile fremitus and vocal resonance lessen, and the percussion note becomes less dull, more tympanitic, and finally resonant (although more or less impairment of the note may persist for a long time). The breath sounds become less bronchial, bronchovesicular, and finally (but often not for a long time) purely vesicular, and coarse and fine bubbling rales, together with in some cases the recurrence of the crepitant rale (so-called redux crepitus), make their appearance. In some cases the physical signs are much delayed, although the diagnosis is quite evident from the symptoms—chill, fever, pain, cough, rusty expectoration, and dyspnœa. In

some cases physical signs are never found. I have known them to be delayed until nearly or about the time of the occurrence of the crisis.

Clinical Varieties.—A number of terms have been employed to designate different varieties of pneumonia. Thus basal pneumonia is sufficiently explanatory of the majority of cases. Apical pneumonia is common in children, more likely to be attended by cerebral symptoms and profound intoxication, and often escapes detection because of the frequent slight cough and expectoration, and because the apices posteriorly are not carefully examined. In adults it is said to be more fatal than the basal form. Double pneumonia is that form in which both lungs, or portions of both lungs, are involved. Migratory, or creeping pneumonia, is that in which different portions of one or both lungs are successively involved. In this the onset may be gradual, the course is prolonged, exacerbations are common, and the disease usually ends by lysis. Massive pneumonia is a form in which an entire lung, or at least a large portion of a lung, is involved and the fibrinous exudate extends from the alveoli to the bronchi. Casts of the bronchi revealing dichotomous divisions may appear in the sputum. The physical signs much simulate those of pleurisy—there being flatness, absence of breath sounds, and of tactile fremitus and vocal resonance. The diagnosis is sometimes suddenly cleared up by the expectoration (by voluntary or involuntary coughing) of obstructing plugs of exudate, and the immediate development of characteristic physical signs. Central pneumonia is that in which the process commences in the centre of a lobe, and, spreading gradually to the surface, does not immediately occasion demonstrable physical signs, and in which the diagnosis, for a time at least, rests upon the symptoms. Latent pneumonia is that which pursues its course unsuspected for some time, or altogether. Many cases of central pneumonia are of this nature, as is also pneumonia in the aged, and many cases developing in the course of Bright's disease, insanity, and alcoholism. In alcoholics, especially, pneumonia is frequently entirely unsuspected, the symptoms being attributed to meningitis, delirium tremens, or uremia.

Epidemic pneumonia, as the term implies, is an epidemic form of the disease. It may be widespread or local. Thus, family, house, jail, ship, etc., epidemics are well recognized. These often have a high mortality rate, and may be associated with influenza. Larval, or abortive, pneumonia is a form of short duration and usually mild symptoms, common especially during epidemics. The initial chill, fever, cough, rusty expectoration, and ill-defined or negative physical signs may be followed by complete recovery in a few days. Alcoholic pneumonia, pneumonia in alcoholics, is usually attended by marked nervous symptoms and delirium, and is often devoid of suggestive local manifestations. It is insidious in onset, has a relatively low fever range (101° to 102° F.), and a very high death rate. Typhoid pneumonia is a pneumonia characterized by marked asthenia or adynamia (asthenic or adynamic pneumonia) or the typhoid state (and has nothing whatever to do with typhoid

infection or typhoid fever). The characteristic of the disease is extreme toxemia (toxic pneumonia)—whence it indicates severity of infection and sometimes double infection (pneumococcic and streptococcic or staphylococcic, bacteremia being common). It must be distinguished from pneumotyphoid—typhoid infection of the lung occurring in the course of typhoid fever. It is also true that pneumococcic infection of the lung may occur in typhoid fever. Bilious or malarial pneumonia is a term used by some writers to designate pneumonia occurring especially in malarious regions, and attended by jaundice, vomiting, and severe nervous symptoms. The complicating symptoms are usually due to an associated duodenal catarrh, and bear no relation to the malarial parasite, although infection with the pneumococcus and the malarial parasite (as well as with other microörganisms) may be concurrent.

Secondary pneumonia is common in many of the acute infective diseases. It may be due to the pneumococcus or to the microörganisms provocative of the primary disorder (typhoid fever, diphtheria, etc.), or to mixed infection with streptococci, staphylococci, and colon bacilli, etc. It is generally bronchopneumonic in type, and devoid of most of the characteristic clinical and anatomical features of true lobar pneumonia. Terminal pneumonia (usually bronchopneumonia) is a common event in the final stage of many cases of Bright's disease, diabetes, carcinoma, heart disease, and the adynamia of senility. The onset is gradual and the symptoms slight and not suggestive; there may be, indeed, few clinical manifestations aside from dyspnœa and slight fever.

Pneumonia in the aged is often latent, and may be terminal. The onset may be sudden, but it is usually insidious, and the local symptoms and physical signs are slight, although the patient is manifestly seriously ill and usually soon dies from cardiac debility. This is a hypostatic or bronchopneumonia, and is especially common in the aged who are confined to bed—as with a fractured femur, for instance. Pneumonia in children and infants often begins with a convulsion or vomiting, the apex is often involved, cerebral symptoms are marked (the disorder much simulating meningitis), expectoration is absent, and the disease usually ends in recovery.

Postoperative and ether pneumonia occurs sometimes after operation and anesthetization, especially after abdominal operations (probably because the bronchial secretions are allowed to accumulate on account of the pain provoked by coughing). The development of the disease is much favored by winter season, by prolonged anesthetization, general debility of the patient, disease of the respiratory tract, such as bronchitis or chronic congestion from heart disease, and by the aspiration of irritating particles (aspiration or inhalation pneumonia). It is often lobular (that is, bronchopneumonia) rather than lobar, insidious in onset, latent in course, and frequently due to microörganisms other than the pneumococcus.

Complications and Sequels.—Inflammations of the serous membrane, pleuritis, pericarditis, endocarditis, and meningitis, are common complications—pleuritis the most common, and meningitis the most serious. In most of these complicating exudates, the pneumococcus may be detected, sometimes alone, sometimes in association with the streptococcus, staphylococcus, etc. Pleuritis is a part of the pneumonic process rather than a complication, since it occurs in all cases in which the inflammation reaches the surface of the lung. In most cases the exudate is slight in amount and fibrinous in character; in other cases it is serofibrinous, and more or less abundant (pleuropneumonia, metapneumonic pleuritis). In still fewer cases, however, but rather commonly in children and youths, the exudate is purulent (metapneumonic empyema): most cases of supposed, unresolved pneumonia. Unusual prolongation of the disease, chills, fever, sweats, leukocytosis, flatness on percussion, markedly increased resistance, absence of breath sounds and of rales, suggest the presence of the pleural lesions; but an absolute diagnosis may require the use of the exploring needle. Pericarditis is a rather unusual complication, more common in left-sided than in right-sided pneumonias. It is usually fibrinous, but may be serofibrinous or purulent. Its presence may be inferred from the development of undue dyspnœa and pericardial distress, but its diagnosis depends upon the detection of the physical signs. Acute endocarditis, both simple and malignant, is more common; it affects the left side of the heart rather than the right, especially if there be an old valvular lesion. The diagnosis rests upon detecting the ordinary manifestations of acute endocarditis—sepsis and embolic phenomena. Acute meningitis, usually fibropurulent, is a rather uncommon, and usually fatal, complication. Frequently it cannot be distinguished from the meningism of many cases of pneumonia without resort to lumbar puncture, which may reveal pneumococci in the meningeal exudate. That the pneumococcic process is a more or less widespread infection in many cases is evident from the occurrence together of inflammation in divers organs, such as the lung, pleura, endocardium, meninges, etc. Rarer complications are bronchitis, pneumococcic synovitis and arthritis, toxic degeneration of the myocardium, gastritis, duodenitis (causing jaundice), enteritis, colitis, nephritis, peritonitis, neuritis, phlebitis, heart clots, etc.

Sequels are rare, inasmuch as most cases result in recovery. In some cases, however, relapse occurs; that is, a few days after the crisis the fever returns and persists for several (two to seven or eight) days. Recurrences are common—one attack seemingly predisposing to others, and eighth, ninth, and tenth attacks being by no means unknown. In some cases resolution is much delayed, and although it may ultimately occur after many days or several weeks, the lung being restored to the normal, fibroid induration (chronic interstitial pneumonia) is a very likely event. In other cases purulent infiltration or abscess (single or multiple), gangrene, or tuberculosis results.

Diagnosis.—In the majority of cases, especially in sthenic subjects, the disease is rarely overlooked—the abrupt onset, initial chill, pain in the side, dyspnœa, cough, rusty expectoration, herpes, leukocytosis, physical signs of solidification of the lung, and the course of the fever, being unequivocal. In debilitated subjects, in those the subject of chronic disease, and in the aged, the disease setting in insidiously may escape recognition. Undue dyspnœa and fever should suggest a physical exploration of the lungs. The association of fever and delirium tremens should lead to the suspicion of pneumonia in an alcoholic subject.

The diseases with which pneumonia is most likely to be confounded are typhoid fever, pleuritis with effusion, meningitis, bronchopneumonia, and acute pneumonic tuberculosis. Cases of acute pneumonic tuberculosis, often said to be the consequence of the engrafting of tuberculosis upon a primary pneumococcic infection, are almost always tuberculous from the onset. In the majority of cases the tuberculous nature of the infection is not recognized until the expected crisis does not occur or tubercle bacilli and elastic fibers are detected in the sputum. The tuberculous nature of the disorder, however, may be suggested by previous ill health of the patient, tuberculosis in the antecedents, a gradual onset, an intermittent and later remittent fever, hemorrhagic-purulent and non-viscid sputum, profuse sweats, rapid emaciation, and the physical signs of cavity formation. In other cases, a later involvement of one apex suggests that the primary lesions (which are more frequent in the upper than in the lower lobe) were tuberculous.

Prognosis.—Pneumonia is a very fatal disease—the mortality rate being from 15 to 40 per cent. The prognosis is influenced by: (1) The age of the patient. Children usually recover; the aged usually die. The mortality rate increases with each decade beyond the second. (2) Race. The disease is more fatal in the negro than in the white race. (3) The previous health of the patient. The death rate is high in those debilitated from any cause. It is almost always fatal in alcoholic subjects. (4) The severity of the infection. The prognosis is bad in patients with marked toxemia, persistently high temperature (105° F. and more), cardiac weakness, and absence of leukocytosis. The extent of the lung involvement, unless it be extreme and bilateral, has very little influence on the severity of the symptoms (the degree of toxemia). (5) The presence of complications renders the prognosis much more anxious, as almost all deaths in previously healthy subjects occur in complicated cases. Death in uncomplicated cases is due to the general effects of the toxemia, or to the special effects of the toxemia on the heart associated with the embarrassing results of increased intrapulmonary pressure, or to heart clot.

Treatment.—Although the patient is not notably infectious to those about him, it is well that he be isolated; the sputum undoubtedly should be destroyed, since it unquestionably is the chief source of infection; and it is wise also, especially if several cases of pneumonia have occurred in the same room or the same bed to disinfect the bed and the room (see page 29).

Our general management of the patient should always be guided by a knowledge of the fact that pneumococcic infection of the lung is self-limited, and that we are apparently unable to shorten it or to influence the crisis. Still we can unquestionably do a great deal for our patients. I have not been able to assure myself that the disease can be aborted, even in its very earliest stages; from time to time I have used the different measures recommended for this purpose, such as venesection, aconite, veratrum viride (to bleed the patient within his own vessels), large doses of digitalis (with a view forcibly to stimulate the heart and re-establish the cardiopulmonary circulation); but, although the patients recovered without untoward symptoms, the disease was not aborted. After the disease has become established, that is, after the first twenty-four hours, these methods of treatment, except bleeding (to be referred to presently), are as harmful as they are irrational.

The patient should be placed in a large, readily ventilated room, and the windows should be kept wide open all the time, so as to provide an abundance of fresh air. When available, as in hospital practice, the best results are achieved by placing the patient out-of-doors, as on a porch, for instance; he should, of course, be well protected by a sufficiency of bed-clothes. The fresh air tends to promote an efficient aëration of the blood, it lessens the cyanosis, invigorates the cardiovascular system, reduces the general intoxication, etc. There can be no comparison between the results attained by affording the patient from the beginning an abundant supply of fresh air, and those that ensue upon the giving of canned oxygen when cyanosis has become extreme and the patient's vitality has been almost if not quite depleted by the intoxication.

In the early stages of the disease, at least, the diet should be largely fluid, that is, largely milk; but the milk may be flavored, as recommended for the typhoid-fever subject, or it may be given as junket, etc.; and there can be no serious objection to egg-white or the whole egg (if well borne), or to meat and vegetable soups thickened with a little flour, powdered rice, or barley, or strained oatmeal gruel, cornstarch, arrow-root, blanc-mange, gelatine flavored with wine, calf's-foot jelly, etc. Care must be taken not to overfeed the patient. After the crisis, milk toast, scraped meat, soft-boiled eggs, potato puree, stewed fruits, etc., may be added, and in a day or two sweetbreads, chicken, etc.

The special indications for treatment are to combat the toxemia, to support the overtaxed heart, to relieve certain symptoms, and to treat the complications as they arise. As yet we have no specific. Anti-pneumococcic serum has not realized the claims originally made for it; nor is this to be expected *a priori*, since the pneumococcic toxin is an intracellular rather than an extracellular toxin—whence the difficulty of producing an efficient antitoxin. I have used the serum in a few cases, apparently with some influence on the temperature, and a slight, though doubtful, influence on the toxemia. Encouraging results have recently followed the use of bacterial vaccines and of extracts of washed leuko-

cytes; but sufficient use has not yet been made of these forms of treatment to determine finally their value.

The pain of the early stages is best relieved by twenty to forty dry cups placed on one or both sides; these not only relieve the pain, but also lessen the dyspnœa. The cupping may be repeated twice or thrice within the first twenty-four hours, and again during the second twenty-four hours, if required. Should this be ineffectual, morphine should be given hypodermicly, since pain itself is depressing to the patient and must be relieved. Dover's powder, deodorized tincture of opium, an ice-bag, a Leiter's coil, a light application of the Paquelin cautery, or strapping the side, also may be used; or one may give every three or four hours a pill containing quinine sulphate, 2 grains (0.13 gram), and powdered opium, $\frac{1}{2}$ grain (0.03 gram). This not only relieves the pain and promotes a feeling of general well-being, but is believed to exert also some antiphlogistic action. I have long since given up the use of poultices, the newer clay poultices as well as the better, though more old-fashioned, flaxseed poultices—with considerable increase in the general comfort of my patients. A cotton jacket may be applied to a child—and will do the mother considerable good. During this stage the blood pressure, if high, may be reduced—by bleeding, free catharsis, and diaphoresis. The abstraction of twenty ounces of blood in sthenic subjects is often followed by relief of pain, dyspnœa, and cerebral (toxic) symptoms; but it should not be practised indiscriminately, and not in debilitated subjects. An initial dose of 10 grains (0.6 gram) of calomel, followed by a saline cathartic, subserves a very useful purpose, promoting free evacuation of the bowels and reducing the blood pressure. If given dry on the tongue and allowed to mix with the secretions of the mouth and throat it exerts also a local antiseptic action. Potassium citrate, solution of ammonium acetate, spirit of nitrous ether, etc., also promote the comfort of the patient by inducing sweating, reducing the blood pressure, and promoting diuresis and elimination of toxins.

The use of hydrotherapy, of water internally and externally, is the best means at our command to combat the effects of the toxemia: given internally, being our best diuretic, it promotes the free discharge of toxin with the urine; applied externally, by its stimulating effects, it counteracts the depressing effects of the toxins. An excellent procedure is to envelop the chest with cloths wrung out of water at a temperature of from 50° to 32° F., to cover these with several thicknesses of dry flannel, and to leave them on for from fifteen to thirty minutes, depending upon the reaction. Often the dyspnœa and the cyanosis are relieved, the pulse becomes stronger, fuller, and less rapid, the manifestations of cerebral intoxication lessen, and the temperature falls to 100° F. or less. The procedure may be repeated every two to four hours, should the temperature again become high and other, especially nervous, evidences of toxemia again become marked. In other cases excellent results follow the application of cloths wrung out of ice water and changed every fifteen minutes for two hours; the treatment may be repeated after an interval of two

hours during the day and of four to six hours at night. The indication for repeating the hydrotherapeutic measures is found in the reaction of the patient. Cool spongings also are sometimes of service. Full tub baths have been recommended, but I have not been convinced of their utility. Warm, full baths, however, are often useful in children. Although the degree of the temperature is utilized as one of the indications for the use of hydrotherapy, the fever, as such, unless very high, really does very little harm, and it is not necessary to reduce it at all hazards; when very high it may be reduced by hydrotherapeutic measures. Under no circumstances whatever should any of the antipyretic drugs be used; they are a distinct menace to the pneumonic patient, reducing his vitality, weakening the heart, lowering the tone of the cardiovascular or vasomotor system, and reducing the oxygenation of his tissues (increasing cyanosis)—only assisting, therefore, the pneumococcic toxin to kill the patient. That they are sometimes given to patients who recover is but one of the many indications of the wonderful resisting powers of the human system.

Whether or not the temperature is high, the patient should be sponged two or three times in the twenty-four hours with cool or tepid water—to promote cleanliness and a general feeling of well-being, to allay nervous irritability and induce sleep; if the temperature is also somewhat reduced this is an additional advantage.

Water internally is of even greater service, since it promotes the free elimination of the toxins. At least three liters should be given to an adult within the twenty-four hours. One of the mild alkaline diuretics, such as potassium bicarbonate, acetate, or citrate, or lemon juice, lime juice, or other fruit juice may be added to it. In the event of renal elimination being inadequate and toxemia increasing, hot enteroclysis and hypodermoclysis (sometimes even intravenous injection) of normal saline solution should be used systematically and repeatedly.

Delirium and other nervous manifestations of toxemia call for stimulants, of which strychnine and alcohol are the best. Strychnine may be given routinely to all patients when toxemia is marked, the heart weak, and renal elimination low; it is wise to begin it early in the hope of averting some of the dangers that threaten. Alcohol had best be withheld until it is required—when it will often tide the patient over the critical period of his disease. From four to eight ounces of whiskey or brandy daily usually suffices. Champagne is excellent for a short time. The more diffusible stimulants, such as cocaine, the aromatic spirit, the carbonate, and other preparations of ammonium, compound spirit of ether, camphor, etc., are useful adjuvants when the heart is failing. Late in the course of the disease, when the heart is dilating from increased intrapulmonary pressure, I have seen excellent results from the use of tincture of digitalis, 8 to 10 minims (0.5 to 0.6 c.c.), combined with 5 to 8 minims (0.3 to 0.5 c.c.) of tincture of belladonna (to increase the vascular tone), every three or four huurs. Digitalin, $\frac{1}{12}$ grain (0.005 gram) hypodermicly, may be substituted for the tincture of digitalis. Caffeine also is of

much value. Atropine hypodermicly is of good service when there is œdema of the lungs and the vascular tone is low; but when vasomotor paralysis supervenes the best results follow the use of hypodermoclysis or intravenous transfusion of physiological saline solution with adrenalin chloride. Finally, when the cyanosis becomes marked, the heart's action feeble and irregular, the muscular element of the first sound of the heart indistinct, and the previously accentuated pulmonary second sound weak, when, in other words, dilatation is marked, good results attend venesection, which may be followed by hypodermoclysis or intravenous injection of normal saline solution with adrenalin chloride. Nitroglycerin is said to be of value when the blood pressure is high, but this is seldom the case. I have never seen much good from its use: indeed, it is usually directly contra-indicated. When the toxemia increases to such an extent as to simulate meningitis (the meningism of the French) lumbar puncture may be practised with hope of good results.

A sufficiency of sleep should be assured the patient—by the mentioned hydrotherapeutic measures, a Dover's powder at night, hot whiskey, a small dose of morphine, or one of the hypnotics. Cough, as such, rarely requires treatment, but in case it is painful and distresses the patient it may be controlled by an occasional Dover's powder, codeine, or heroin. Expectorants, as a rule, are not indicated, usually not until the stage of resolution, when, if the sputum is viscid and tenacious, ammonium chloride, iodide, or carbonate, terpene hydrate, terbene, and similar drugs may be used. Should tympany develop it must be relieved immediately—since it adds to the already existing embarrassment of the heart and lungs; the diet must be regulated, milk stopped, the bowels opened freely, eserine, $\frac{1}{100}$ to $\frac{1}{50}$ grain (0.00065 to 0.0013 gram) given hypodermicly, and efforts made to increase the usually low blood pressure.

The complications and sequels are to be treated on general principles. General tonics, iron, quinine, strychnine, cod-liver oil, phosphorus, etc., are valuable during convalescence. In delayed resolution, recourse may be had to these general measures, and to counterirritation (iodine, blisters, the Paquelin cautery), the iodides internally, and perhaps the cautious use of the *x*-rays, which although not free from danger, are said to foster resolution. Pilocarpine has been recommended, but it is doubtful if it does any good.

MENINGOCOCCIC INFECTION; CEREBROSPINAL FEVER.

(*Epidemic Cerebrospinal Meningitis; Spotted Fever.*)

Cerebrospinal fever is an acute, specific, infectious, and slightly contagious disease caused by Diplococcus intracellularis meningitidis (Weichselbaum); it is characterized anatomically by fibrinopurulent inflammation of the pia-arachnoid, and clinically by irregular course, fever, and divers nervous symptoms.

Etiology.—Cerebrospinal fever, though it occurs at all ages and in both sexes, is more common in children and young adults, and in the male sex. It occurs both epidemically and sporadically, especially in the winter and spring; epidemics are usually circumscribed, rarely widespread, are often more common in country villages and towns than in larger cities, and are much favored by overcrowding and general insanitary conditions (institutions, barracks, jails, etc.).

The exciting cause of the disease is Diplococcus intracellularis meningitidis (or meningococcus), which is found in the cerebrospinal exudate, and sometimes in the nasal secretions and the blood. The disease does not appear to be especially contagious. How the contagium is spread is not definitely known, but acute and chronic meningococcic "carriers" seem sometimes to be the active factor. Fomites are not unduly active. Infection is believed to be acquired through the nose (infecting the meninges through the cribriform plate of the ethmoid bone or through the sphenoidal cells); doubtless the ear is sometimes the portal of entry, and possibly the blood is sometimes primarily involved (by way of the tonsils, etc.). Recurrences are not uncommon; one attack does not confer immunity.

An infection almost if not quite identical, anatomically and clinically, with cerebrospinal fever may be caused by the pneumococcus, streptococcus, staphylococcus, influenza bacillus, etc., but these are usually secondary infections.

Pathology.—In the early stages there is intense hyperemia of the pia-arachnoid; in fulminant cases these may be the only lesions observed. In the ordinary acute cases, there is sero-fibrino-purulent inflammation of the pia-arachnoid, especially marked at the posterior basic region of the brain, and the dorsal surface of the thoracic and lumbar cord; but the exudate may extend to the convexity of the brain, and indeed involve the entire pia-arachnoid. The cranial nerves (especially the second, third, fourth, fifth, sixth, seventh, and eighth) are often embedded in a thick, fibrino-purulent exudate, whereby irreparable damage results. In more chronic cases the pia-arachnoid is irregularly thickened, the ventricles are distended with turbid fluid, and irregular foci of hemorrhage, softening, and encephalitis are encountered. Microscopically the exudate consists largely of polynuclear leukocytes, which contain the meningococcus and certain unusually large endothelial cells; there is also leukocytic infiltration of the brain and cord, foci of hemorrhage and suppuration, and swelling of the ganglion and neuroglia cells. The lesions common to most toxemias are found in the other organs.

Symptoms.—The period of incubation is probably short—one to four days. Usually prodromes are slight—malaise, headache, pains in the back, etc.; but they may be entirely absent. The onset of the disease is commonly abrupt, with severe chill (sometimes chilliness), fever (102° to 103° F.), severe headache, vomiting, soon followed by pain and stiffness in the muscles of the neck. Evidences of general involvement of the nervous system are soon manifest in increasing headache, vertigo,

vomiting (sometimes intractable), delirium, which in the early stages may be wild and maniacal, but with increasing exudation gives way to stupor and finally coma. Involvement of the special senses is seen in early photophobia and intolerance of noises, followed by paretic phenomena—dimness of vision and deafness. Evidences of motor irritability are always seen, and consist especially of spasm of the muscles of the neck (painful retraction of the head) and of the spine (opisthotonos), increased reflexes, tremors and clonic and tonic spasms of other muscles, of which Kernig's sign is an important evidence—inability to extend the leg when the thigh has been flexed upon the abdomen; this, which may be readily accomplished in health, is rendered impossible in most cases of meningitis by the tonic spasm of the flexors of the leg (the ham-string tendons). The ocular and the facial muscles also may be implicated in the spasms. Later, evidences of paresis or paralysis become manifest; these, while they may be present in the extremities (monoplegia, paraplegia, etc.), are commonly most marked about the eye (ptosis, unequal pupils, strabismus, etc.) and the face. Evidences of sensory irritability are found in the general cutaneous hyperesthesia, tenderness of the skull and along the spine, and sometimes of the peripheral nerves. Anesthesia sometimes replaces the hyperesthesia.

The fever is irregular—remittent and intermittent, sometimes subnormal, sometimes very high (105°, 106°, even 108° F. or more just before death). The pulse is variable; it is often accelerated at first, especially in children, but in many cases, especially in adults, it soon becomes very slow—60, 40—and irregular, and it varies much from time to time. The respirations are often increased at first; soon they become normal (except in the event of pneumonia or bronchitis); later they may take on the Cheyne-Stokes type. Herpes facialis occurs in about 60 per cent. of the cases. Petechiæ (whence the name spotted fever) occur in somewhat more than one-half of the cases in most epidemics; in sporadic cases they are often missed; and when present may be few or many (universal). Trousseau's tache cerebrale (persistence of a red line after striking the skin) can usually be elicited. Erythema, urticaria, and even gangrene have been observed. The abdomen is often retracted. Leukocytosis (25,000 to 40,000) is usually present, and is polynuclear in type. Slight albuminuria, rarely glycosuria, may be encountered. The bowels are usually constipated. The spleen may be enlarged, but often is not demonstrably so.

Varieties.—The following types may be recognized: Abortive cases, in which there are severe initial symptoms, which, however, soon subside, and the patient convalesces. Slight cases, in which there is moderate fever of slight duration, ill-developed cerebrospinal symptoms of short duration, and a rather quick and satisfactory convalescence. Malignant or fulminant cases, in which the symptoms are of great severity, and the temperature very high (108° F. or more), and in which death results within thirty-six or forty-eight hours. Apoplectiform cases, in which the patient suddenly becomes unconscious, possibly paralyzed, and soon dies.

Intermittent cases, in which the fever and the other manifestations vary much from time to time, remit and intermit. Chronic cases, in which the disease lasts for months, relapses and improvements occurring from time to time.

Complications and Sequels.—Complications and sequels are many and various. Pneumonia, pleuritis, pericarditis, arthritis, nephritis, and parotitis are not infrequent during the height of the disease. The most common sequels are optic neuritis and atrophy, parotitis, iritis, and panophthalmitis, leading to blindness; otitis media and mastoiditis, leading to deafness, and when occurring in young subjects to deaf-mutism; paralysis of the different cranial nerves, aphasia, hydrocephalus, monoplegia, hemiplegia, etc.

Diagnosis.—The diagnosis is usually evident from the foregoing symptoms. Variability in the intensity of the symptoms from time to time is one of the most characteristic phenomena of the disease. With a view to confirm the diagnosis, lumbar puncture should be resorted to —a three-inch needle with a good-sized caliber being introduced into the fourth intervertebral space (just below a line drawn between the crests of the ilium). Cytodiagnosis may be resorted to: in cerebro-spinal meningitis the polynuclear cells predominate in the exudate, whereas in tuberculous meningitis, the mononuclear cells predominate. Diplococcus intracellularis meningitidis is also easily demonstrated in the exudate by microscopic examination; cultural methods and inoculations into lower animals will help to a diagnosis in doubtful cases. Usually not much difficulty is experienced in differentiating typhoid fever and pneumonia; but in some cases with marked meningism the differential diagnosis may be for the moment impossible without resort to lumbar puncture, and since this is not only a diagnostic measure, but also a therapeutic measure of value, it should not be delayed. Meningitis, of course, may complicate pneumonia and typhoid fever.

Prognosis.—The mortality varies much in different epidemics—from 25 to 75 per cent.—and is usually in direct ratio to the severity of the cerebral symptoms. Fulminant cases may result fatally within twelve hours; and ordinary acute cases in from four to ten days. In favorable cases convalescence usually sets in at the end of ten to fifteen days, but it may be much delayed, and death may eventually result, generally from complications or exhaustion, at the end of weeks or even months.

Treatment.—Isolation and other prophylactic measures (page 29), rest in bed, quiet, and a liquid and soft nutritious diet comprise the general measures useful in the disease. Flexner's antimeningococcic serum, although still on trial, seems to be more or less of a specific. In using it, lumbar puncture should be first performed, and then 30 c.c. (in fulminant cases, 45 c.c.) of the serum should be injected. This should be done daily for four or five days; more frequently, if the symptoms are alarming, and for a longer period, if the symptoms persist. The use of extract of washed leukocytes has been suggested by Hiss. In the absence of a specific, treatment is largely symptomatic. Hydro-

therapy is of value in most cases, and in addition to a tub bath, should the temperature be high, much relief is afforded by an ice-bag to the head and along the spine. Hot baths are often grateful to the patient, and seem to lead to some improvement in the general symptoms. Counter-irritation by means of the Paquelin cautery is much preferable to blisters. Preparations of opium are useful in relieving the headache, the pains in the back, and the muscular contractions, and may often be serviceably combined with atropine or the bromides. Potassium iodide, ergot, quinine, salicylic acid, cannabis indica, and mercury (for its antiseptic properties) have also been recommended. Pilocarpine has been reported of value by reason of the leukocytosis it induces. Stimulants may be used when exhaustion supervenes. Lumbar puncture is a very serviceable procedure—not only diagnostically but therapeutically. The withdrawal of 20 to 30 c.c. of cerebrospinal fluid is often followed immediately by relief of the headache and the muscular contractures, and it is reasonable to suppose that a certain amount of toxins is also withdrawn. The procedure may be repeated from time to time. It has been suggested to irrigate the cerebrospinal canal with physiological saline solution; perhaps with improvement in technique this may become a well-indicated therapeutic measure.

GONOCOCCIC INFECTION.

Gonococcic infection is an acute, specific, infectious, and highly contagious disease caused by Micrococcus gonorrhœæ (Neisser), and characterized by blenorrhagia and sometimes by general systemic infection.

Etiology.—The specific cause of the disease is the gonococcus, which, although usually acquired in sexual intercourse and attacking first the mucous membrane of the urethra in the male, and the vulva, vagina, and urethra in the female, may nevertheless attack any mucous membrane, such as the conjunctiva, the anus, the oral mucous membrane, etc. While the lesions that it provokes are often confined to the genito-urinary tract, it not uncommonly causes the most widespread local disease, such as cystitis, prostatitis, vesiculitis, epididymitis, orchitis, etc., in the male, and endocervicitis, metritis, salpingitis, ovaritis, peritonitis, etc., in the female; it is one of the most prolific causes of continued ill-health in both sexes, but especially in women the innocent victims of uncured, more or less quiescent, antemarital infection in their husbands; and it sometimes gives rise to general infection. The general manifestations of the local infection are due, in the great majority of cases, to toxemia; occasionally, however, to gonococcic bacteremia. Although arthritis, endocarditis, pericarditis, myocarditis, myelitis, keratitis, iritis, and panophthalmitis, etc., have been reported, special importance in the present connection attaches to: (1) Gonococcic bacteremia; (2) gonococcic endocarditis; and (3) gonococcic synovitis and arthritis.

Symptoms.—*Gonococcic bacteremia* (septicemia, pyemia) has occasionally been observed in association with a focus of suppuration in the

genito-urinary tract, and with gonococcic endocarditis and arthritis. The symptoms have been those of profound intoxication together with the manifestations of the local infection. The gonococcus has been cultivated from the blood.

Gonococcic endocarditis has been observed in a few, but gradually increasing number of cases. It has usually been associated with (sometimes preceding, sometimes following) arthritis, and the gonococcus has been isolated from the blood; in other words, the condition is a bacteremia. The symptoms do not differ from those of endocarditis due to other causes. Pericarditis and myocarditis have been observed in a few fatal cases.

Gonococcic synovitis and arthritis develop usually when the acute urethritis is subsiding, occasionally when it has become chronic. It is somewhat more common in men than in women. It may involve one or several joints; more than one are more frequently involved than is generally supposed. The knee (men), wrist (women), and ankle are most likely to be attacked, but the smaller joints are not infrequently affected. Irregular fugitive pains and possibly some tenderness are common during many attacks of gonococcic urethritis, but they are doubtless expressions of the toxemia and they soon subside. In other cases the more common manifestations of synovitis develop—extreme pain, tenderness, swelling, and inability to move the joint. Slight fever may be present for several days, but in most cases (in the absence of suppuration) it soon subsides, and the general symptoms are inconspicuous as contrasted with the local. The effusion is within and without the joint, and may extend along the fasciæ and the tendons and involve the bursæ. It is usually serous or serofibrinous in character. By aspiration and culture methods the gonococcus may be recovered, but a mixed infection is not unknown. Occasionally suppuration results (suppurative synovitis, empyema or phlegmon of the joint) and may result in widespread destruction of the articulating surfaces. Mild infections may result in recovery, but permanent stiffness (fibrous ankylosis) due to chronic inflammation of the articular and periarticular structures is much more common. Occasionally a chronic hydrops (hydrarthrosis) ensues. Bony ankylosis may result from the more destructive infections. Exostosis, especially of the os calcis, is not uncommon, and gives rise to local pain, tenderness, and a "tiptoeing gait" (one form of "painful heel").

Diagnosis.—Gonococcic synovitis and arthritis may be distinguished from rheumatic fever (to which it is in no way related) by the history of urethritis, severity of the local lesions, relative meagreness of the general symptoms, persistence of the articular lesions and their nonresponse to the salicylates, and the more likely involvement of the temporomaxillary, sternoclavicular, sacroiliac, pubic, and intravertebral joints.

Treatment.—The urethritis, whether acute or chronic, should receive attention. The salicylates and potassium iodide are of very little, if

any, service. Some good seems to attend the use of syrup of iron iodide, 10 to 60 minims (0.6 to 4 c.c.) three times a day. In most cases tonics are called for sooner or later, in which event iron, quinine, strychnine, arsenic, and cod-liver oil may be valuable. For the relief of the local symptoms nothing is as efficacious as absolute rest enjoined by a fixed dressing. All other measures, such as hot and cold applications, lead water and laudanum, counterirritation, ointments of ichthyol, belladonna, mercury, iodine, etc., though of value, are of subsidiary importance. If the joint has become fixed in a poor position, it must be straightened, under an anesthetic, if necessary. If improvement is not manifested soon, the joint should be opened, irrigated, and subsequently treated aseptically. Counterirritation (blisters, thermocautery), massage, passive movements, superheated dry air ("baking the joint"), and Bier's "passive hyperemia" treatment are sometimes of value in relieving some of the chronic manifestations. An antigonococcic serum has been used, with indifferent success. Bacterial vaccines seem to have been of service in some cases.

TUBERCULOSIS.

(*Phthisis; Consumption; Scrofula.*)

Tuberculosis is a specific, infectious, and mildly contagious disease caused by Bacillus tuberculosis (Koch); it is characterized anatomically by the development of small nodules, so-called tubercles, or a diffuse productive inflammation, both of which tend to undergo caseation and softening or, less commonly, fibrosis and calcification; the clinical manifestations vary, depending upon the type of infection and the tissues especially involved.

Etiology.—Tuberculosis is an exceedingly widespread disease; it occurs in all parts of the world, although it is most prevalent in the temperate zone and at low rather than high altitudes; and it affects many species of animals, notably man, cattle, fowls, birds, domestic animals, etc. Of human subjects, no race is exempt; the negro and the American Indian display a high degree of susceptibility, and the Hebrew apparently some relative immunity. The disease occurs at all ages, but there seems to be a special susceptibility during the first two years of life (perhaps due to milk-borne infection or close association with a tuberculous mother), and during adolescence and early adult life (fifteenth to twentieth years). Sex as such is without etiological significance; the frequency of the disease in women may be in part attributed to a sedentary indoor life, lack of outdoor exercise, pregnancy, and lactation—which reduce the resisting powers. The poor and the industrial workers are especially subject to the infection; both often are provided with insufficient and improper food and clothing; they live and work in dark, overcrowded, ill-ventilated, and otherwise insanitary rooms; and they are subject often to prolonged and exhausting labor and to the inhalation of irritating, dust- and bacilli-laden atmosphere, etc. Other predisposing

factors comprise trauma (especially of the bones and joints); catarrhal inflammation of the respiratory tract (often tuberculous from the beginning); infectious diseases like pneumonia (often tuberculous from the beginning), influenza, pertussis, measles, typhoid fever, syphilis, etc.; debilitating diseases, such as diabetes, carcinoma, etc.; aneurysm and compression of the lung, and chronic diseases of the heart associated with anemia of the lungs, such as aortic valve disease: mitral valve disease, in consequence of the chronic pulmonary congestion that it induces, seems to afford some immunity to tuberculosis of the lungs. Some persons seem to possess a more or less efficient natural immunity, some of these being descendants of tuberculous ancestors. One attack of the disease does not confer immunity, at least not a lasting immunity, since recurrences or relapses are the rule.

The exciting cause of the disease is Bacillus tuberculosis, of which four types may be distinguished—the human, the bovine, the avian, and the piscian. These, perhaps, originally comprised a single type, but owing to environmental conditions have developed special individual characteristics. The human and the bovine types are intercommunicable, but the avian and the piscian types appear to be either incapable of producing tuberculosis in man or capable of producing it only in a mild form. Raw has recently suggested that in the human subject the human type of the bacillus affects the lungs, the intestines, and the abdominal viscera, while the bovine type affects the lymph nodes, the peritoneum, the meninges, the bones and joints, and probably the skin—an opinion that has not yet been confirmed. Infection is transmitted largely through the medium of infected air or infected food; but it may be transmitted also by direct contact or inoculation, and perhaps by a third person, by fomites, and by domestic animals. The air becomes infected through the drying and subsequent dispersion of bacilli-laden sputum, and through the projection into the atmosphere by coughing of minute, invisible, droplets of infected sputum and saliva. Food, especially milk and butter, becomes contaminated by tuberculosis especially of the udders of cattle, or by being incautiously handled by tuberculous subjects; the meat from tuberculous cattle also may be infected by widespread tuberculosis of the animal; and the milk, meat, butter, and other foodstuffs may become infected by flies and other insects that have walked on or fed upon tuberculous sputum. Infection may be acquired: (1) By inhalation—which is doubtless the most common mode of infection. The bacilli probably gain access to the tissues through the walls of the bronchi or bronchioles, and set up a primary pulmonary tuberculosis. (2) By ingestion—probably the second most common mode of infection. The bacilli carried in foodstuffs may penetrate the intestinal mucosa without producing discoverable lesions; thence they gain access to the mesenteric lymph nodes, where they may set up tuberculous lesions; or, according to some observers, they invade the thoracic lymphatics and from these subsequently involve the lung, giving rise in this manner to pulmonary tuberculosis. (3) By way of the tonsils and

other lymphatics of the oral and pharyngeal cavity (such as carious teeth, etc.). Tuberculosis of the cervical lymph nodes ensues, and from these foci, dissemination may occur to other parts of the body, especially the lungs. (4) Direct inoculation into the skin through an abrasion, as may occur at dissections, operations, necropsies, etc.; the ensuing lesion is often spoken of as an anatomical tubercle. (5) By direct inoculation of the genitalia in sexual intercourse—a rare mode of infection, but perhaps responsible for some cases of tuberculosis of the uterus and Fallopian tubes; rarely of other parts of the genitalia in both sexes. (6) By way of the placenta—the fœtus becoming infected by a tuberculous mother. This so-called congenital tuberculosis is very rare in the human subject; it seems to be more common in certain of the lower animals, especially cattle.

Infection having occurred, the bacilli may be disseminated more or less widely throughout the body: (1) By way of the lymphatics. This is the commonest mode of dissemination, especially in all forms of tuberculosis in children, and in the subacute and chronic pulmonary, intestinal, and other types of the disease in adults. (2) By way of the bloodvessels. This is the mode of dissemination in miliary tuberculosis, the bacilli gaining access to the vascular channels through rupture thereinto of a tuberculous focus, most commonly the rupture of a tuberculous bronchial lymph node into a pulmonary vein. (3) By way of a mucous surface. This is a common mode of extension of the lesions in pulmonary tuberculosis, infectious material (sputum) being discharged into a bronchus and then aspirated into another bronchus that leads to an uninvolved portion of the lung; certain intestinal ulcers also perhaps exemplify this mode of dissemination, sputum being swallowed and infecting the intestinal mucosa.

Pathology.—Although no organ of the body is exempt from tuberculosis, the incidence of involvement varies considerably. In children, the lymph nodes, bones, and joints are most commonly involved; in adults, the lungs, intestines, urinary organs, serous membranes, etc. The lesions consist of two main types—tubercle formation and a diffuse productive inflammation. The characteristic lesion is the tubercle, which in its earliest stage, the so-called miliary tubercle, consists of a grayish, somewhat translucent nodule, a millimeter or less in diameter (the size of a millet seed); it is firmer than the surrounding tissue, projects slightly from the general level of the cut surface of an organ, and may be delimited by a narrow zone of reactive congestion. In the course of time these miliary, or gray, tubercles increase in size by peripheral extension, and concurrently therewith their centres become yellowish in color and soft—the so-called yellow, or caseous tubercle. In consequence of the coalescence of neighboring tubercles, large tuberculous masses form and may attain the size of a hen's egg (the so-called solitary tubercle, common in the brain and meninges); and in consequence of central caseation and liquefaction necrosis, a so-called tuberculous (or cold) "abscess" (consisting, however, only of more or less fluid or inspissated

granular debris) results; this being discharged externally leaves a cavity (especially common in the lungs), or one or several sinuses (especially common in connection with bones and joints). In the neighborhood of coalesced tubercles, small gray or yellowish tubercles are usually encountered, the result in most cases of lymphatic extension (so-called mother and daughter tubercles). The much rarer diffuse productive inflammation may occur in any organ, but is most common in the lungs and the lymph nodes. It may or may not be associated with the formation of definite tubercles. For instance, the inhalation of large numbers of virulent bacilli by susceptible individuals may lead to the rapid development of tuberculous pneumonia, which exhibits many clinical and pathological analogies with ordinary pneumococcic infection of the lung. In the early stages, however, the lung has a more gelatinous appearance, due to certain peculiarities of the inflammatory œdema; later it becomes cheesy in character, and often the seat of widespread softening and excavation.

The first effect of tubercle bacilli settled and multiplying in the tissues appears to be irritative rather than degenerative—proliferation rather than death of the cells. The earliest lesions consist of the development of large, ovoid, or polygonal cells with ovoid or elliptical, vesicular nuclei, and an abundant cytoplasm, harboring usually few or many tubercle bacilli. These cells, spoken of as epithelioid or endothelioid, on account of obvious resemblances, are derived from proliferation of the endothelium of capillary bloodvessels and lymphatics, as well as other fixed connective tissue cells. About this focus of epithelioid cells (so-called epithelioid tubercle) leukocytes collect in greater or less numbers: at first many polynuclear cells, but soon mononuclear cells predominate (so-called lymphoid tubercle). This cellular accumulation, which does not differ from the small round-cell accumulations of inflammation, comprises lymphocytes from the surrounding vascular and lymphatic channels, as well as proliferated fixed connective cells; that is, the chemotactic influence of the tubercle toxin seems to be lymphocytic rather than polynuclear leukocytic. About this time, the capillaries of the tuberculous focus become occluded, and since new capillaries are not formed, the tubercle soon becomes both extravascular and avascular. In its early stages the tubercle may consist only of these epithelioid and lymphoid cells, in varying proportion, and a delicate reticulum, doubtless the remains of stretched and otherwise altered fibers of the fixed connective tissue. In many cases, however, so-called giant cells also are encountered: large, irregular cells, with many nuclei arranged peripherally or toward one pole, and containing many tubercle bacilli. These result from fusion of neighboring epithelioid cells or from nuclear proliferation (partly amitotic) without coincident cytoplasmic division. The appearance within these giant cells of a hyaline or granular centre, as well as certain peripheral extensions, suggesting perhaps fusion of adjacent epithelioid cells, indicates that the giant cell is the first retrograde change within the tubercle. Soon, however, other evidences of necrosis

supervene—the result of the direct action of the tubercle toxin as well as the avascularity: the nuclei of the central epithelioid cells undergo various retrograde changes (hyperchromatosis, karyolysis, karyorrhexis, pyknosis), the cytoplasm becomes granular (ground-glass appearance), and the cell outline becomes indistinct; ultimately all cellular structure disappears, and the central portion of the tubercle becomes converted into a granular cheesy mass. This necrosis increases by peripheral extension, so that ultimately the tubercle consists of a central necrotic area, and more or less ill-defined zones of epithelioid cells and giant cells, and of round-cell infiltration. In some cases, in consequence of immunizing processes, caseation becomes retarded or arrested, the peripheral fibroblasts are enabled to build up more or less efficient new connective tissue, which may or may not contain tubercles, and ultimately the tuberculous focus may be surrounded, encapsulated, or pervaded by fibrous connective tissue (fibrocaseous tubercle). This fibrosis may progress until the tuberculous focus has been completely absorbed, and cure ensues; more commonly, perhaps, more or less caseous material remains within the connective tissue capsule, and the capsule itself may become calcified (quiescent, inactive tuberculosis). In the event of progressive caseation, secondary infections are quite likely to ensue, especially in the lungs—infections with the pneumococcus, staphylococcus, streptococcus, influenza bacillus, Micrococcus tetragenus, etc. The general toxemia that results from the absorption of the tubercle toxin leads to fever, emaciation, amyloid and fatty degeneration, etc.; but some of the general manifestations, such as the so-called hectic fever, are due in part to the toxemia of the secondary infections.

Acute Miliary Tuberculosis.

Etiology.—In addition to the general etiological factors, already mentioned, special importance attaches to the predisposing influence in miliary tuberculosis of : (1) Childhood and youth, perhaps accounted for by the great activity of the lymphatic system, especially its active absorption and conduction; (2) localized chronic pulmonary tuberculosis, in which the fatal termination is frequently determined by dissemination of the process (miliary tuberculosis)—in the lungs or more or less widely throughout the body; (3) other acute infections, such as pertussis, measles, typhoid fever, etc., which are not infrequently followed by miliary tuberculosis; (4) trauma, the mechanical effect of which may be to permit the entrance of tubercle bacilli into the blood stream, or to activate a quiescent tuberculous focus which later may lead to dissemination of the infection; (5) pregnancy, the puerperium, and lactation, which may reduce the resisting powers; and (6) debilitating diseases, like diabetes, cirrhosis of the liver, chronic nephritis, etc., in which the lethal issue is often due to miliary (and other forms of) tuberculosis.

Pathology.—Acute miliary tuberculosis may be confined to one or several organs, or it may be widespread through the body. The infection

is transmitted by the blood stream, to which the bacilli gain access in consequence of, (1) the irruption of a tuberculous focus into the vascular stream, or (2) tuberculous disease of the wall of a vein, artery, or lymph vessel. The characteristic lesion consists of the miliary or gray tubercle; but in some cases, of comparatively slow progression or moderately long duration, more or less caseation, the so-called yellow tubercle develops. These tubercles, especially the miliary or gray tubercle, may be found in any organ or tissue of the body, but they are most common in the lungs, liver, spleen, kidneys, intestine, meninges, etc. If, as is common, there is primary disease of the lungs or the peribronchial lymph nodes, the bacilli may gain entrance into the pulmonary veins, and be thence transmitted to the heart and the systemic circulation. In other cases, the bacilli may gain the blood stream by way of the thoracic duct. In cases of disseminated infection, tuberculous arteritis, phlebitis, and lymphangitis have been found, but it is not always possible to determine whether these vascular lesions are the result or the cause of the generalized disease; older lesions in the vessel walls, however, suggest that they are the antecedent process. Tubercles have been found in the walls of the thoracic duct, the pulmonary veins, the jugular veins, the aorta, and certain smaller arteries, etc. In the lungs miliary tubercles are usually present in greatest numbers, and are likely to be most widespread in distribution; they may develop in the alveolar walls or in the peribronchial or perivascular tissues. The liver is almost always affected in generalized miliary tuberculosis; two sources of infection are possible—the portal and the general arterial circulatory system; the tubercles may be found in the intralobular or interlobular tissue, and there may be also some hyperplasia of the connective tissue and proliferation of the biliary ducts. In the spleen the tubercles may be distributed regularly or irregularly. In the walls of the intestine tubercles are common, especially in the submucous and serous coats, and they are usually associated with adjacent ulceration. Infection may occur through the lumen of the gut or by way of the blood stream. In the kidneys, which are commonly involved, the tubercles are most frequently seen in the cortex. The serous membranes, notably the cerebrospinal meninges, the peritoneum, the pleuræ, and the pericardium, are commonly involved; occasionally also the endocardium. The tubercles develop also in the bone marrow, the choroid coat of the eye, etc., but they seem to be comparatively uncommon in the pancreas, thyroid body, the myocardium, the stomach, the uterus, etc.

Symptoms.—The symptoms vary considerably in different cases. In the one series of cases, following a more or less obvious tuberculous focus somewhere in the body (lungs, lymph nodes, testicles, bones, joints, etc.), evidences of general involvement of the body develop—the so-called general, typhoid, or toxic form; in another series, the major symptoms are referable to the lungs—the so-called pulmonary form; and in still another series, the chief phenomena are cerebral—the so-called meningeal form; but in most cases the lesions are more or less widespread,

although the predominant symptoms may be referable to one or another system of organs.

In cases without an obvious antecedent tuberculous focus somewhere in the body, the first noteworthy symptoms consist, as a rule, of general failure of the health, weakness, anorexia, and fever. The fever may be continuous in type, or from time to time remittent.

In pulmonary infection, the sudden development of high temperature running continuously over an extended period is always suggestive of the development of miliary tuberculosis. The irruption of the infectious material into the blood doubtless marks the inception of the pyretic condition. The temperature may reach 104° or 105° F.; there is usually a drop of 1° or 2° in the morning temperature, but now and then this may be reversed, the evening temperature being the lower, which by some observers has been held to be diagnostic of the disease. Although the majority of cases present fever of one or another type, some show a continuously subnormal temperature; some of these may be interpreted as cases of cerebral infection, in consequence of which depression of the thermogenic centres has taken place. On the contrary, others show no such involvement, the absence of fever seeming to be an expression of insensibility of the heat-producing centres.

Certain respiratory symptoms, such as dyspnœa, cough, and cyanosis, are quite constant. Dyspnœa may be intense, especially in children, a respiratory rate of 75 to 90 per minute not being unusual. This is produced not infrequently without especial lung involvement, in which event it has been ascribed to a toxic condition of the respiratory centres and to the development of tubercles on the diaphragm. Miliary tubercles in the pulmonary tissues may be sufficiently irritating to exert a pronounced effect upon the pneumogastric nerve. Cheyne-Stokes breathing is not infrequently observed. Dyspnœa and increase of pulse rate are usually coincident in their development, but not rarely dyspnœa has the precedence. Cough is an important symptom, preceding not infrequently the development of dyspnœa. It is probably due to the toxemia, as well as to the mechanical effect of the tubercles in the lungs. It is variable in its intensity, some cases presenting a violent paroxysmal cough which, when associated with severe dyspnœa, simulates asthma. In the early stages of the affection there is little, if any, expectoration, but now and then small quantities of blood may appear. This rarely is in such quantities as to resemble the rusty sputum of croupous pneumonia. In the later stages, the expectoration is more abundant and becomes mucopurulent in character. This picture is somewhat altered when there has been an antecedent chronic pulmonary tuberculosis, and the acute generalized condition is a complication. Under these circumstances the dyspnœa and cough usually have existed for an extended period, and there is usually an exaggeration of the symptoms upon the development of the additional infection. Cyanosis may be extreme, more so than in most other diseases, and is quite significant of

miliary tuberculosis of the lungs (eliminating emphysema and pneumonia).

The cardiovascular system is more or less affected. The heart action becomes accelerated, the sounds become weak, and passive congestion and œdema of the dependent parts mark the later stages of the disease. The blood pressure is typically lowered. The pulse is quickened, averaging 120 to 150 beats per minute, and at times is dicrotic in character. The greatest acceleration of pulse rate is seen in children, while in the aged it may be below 100 per minute.

Anorexia is constant, and nausea and vomiting are often present, especially in meningeal infection. Abdominal tenderness and meteorism are at times noted, especially in the presence of peritonitis. The patient is usually constipated, though diarrhœa may occur from time to time, and be associated with hemorrhage, with or without intestinal ulceration. The amount of urine excreted is usually small, the result in part of profuse sweating, especially at night. In practically all cases there is a low-grade nephritis which accounts for a slight albuminuria. Peptonuria and excessive excretion of urates often accompany the disease.

The skin generally is pale, but in the peripheral portion of the body, as the fingers and toes, more or less, sometimes extreme, cyanosis is evident. Eruptions of several types are sometimes seen, such as herpes, petechiæ, miliaria, sudamina, etc. Miliary tubercles are rare cutaneous lesions. Profuse sweating occurs in practically all cases, and is due to a toxic effect upon the nervous system. This generally appears at night and is coincident with a fall of temperature.

Certain nervous symptoms are seen in almost all cases of generalized miliary tuberculosis without meningeal involvement, such as headache, vertigo, hebetude, and somnolence; they are referable to the toxemia. The headache may be mild or severe, frontal or vertical in type. It is usually most marked during the periods of increment of fever. Vertigo is usually not so conspicuous unless there is meningeal infection. Hebetude and somnolence are often marked in the later stages. When meningeal infection is present the nervous symptoms are intense and tend to overshadow the general symptoms, such as cough, dyspnœa, profuse sweating, etc. Convulsions, paralyses, vomiting, painful contracture of the posterior cervical muscles, strabismus, contraction or dilatation of the pupils, and flexion-contracture of the knee (Kernig's sign) may occur. Delirium, coma, and death often terminate the condition. Tubercles may develop in the choroid coat, usually of both eyes.

Physical Signs.—The physical signs, as a rule, are merely those of bronchitis, although, of course, in some cases they are modified by the signs of antecedent pulmonary tuberculosis, such as solidification or cavity formation. The percussion note may be somewhat hyperresonant —from inflation of the lungs. The breath sounds are harsh and the expiration prolonged; the rales may be fine crackling or bubbling, or sibilant and sonorous; frictions may be heard (pleural tubercles). In the course of time, a soft systolic murmur may become audible at the

apex of the heart, and the pulmonary second sound may become accentuated (congestion of the lungs). The liver and the spleen are sometimes moderately enlarged from congestion and the presence of tubercles; they are rarely tender. Meteorism, abdominal tenderness, and rigidity are often seen in involvement of the peritoneum.

Diagnosis.—The pulmonary form of miliary tuberculosis is usually readily recognized from the detection of an antecedent tuberculous focus in the lungs or elsewhere, the presence of cough, dyspnœa, marked cyanosis, the physical signs of bronchitis, and tubercle bacilli in the sputum (which, however, may be missed until late in the course of the disease). Cerebral symptoms suggest meningeal involvement, which may be determined definitely by finding tubercle bacilli and an excess of lymphocytes in the cerebrospinal fluid, or by detecting choroid tubercles. The general, toxic, or typhoid form resembles and is commonly mistaken for typhoid fever, but its likelihood is suggested by absence of the characteristic typhoid fever curve, of abdominal symptoms, of erythematous rose-spots, of leukopenia, and of the Gruber-Widal reaction, and by the presence of increased pulse rate, of dyspnœa, of cyanosis, of occasionally bloody sputum, of choroid tubercles, and of tubercle bacilli in the cerebrospinal fluid and rarely in the blood.

Prognosis.—The outlook is extremely grave; so far as is known the termination is invariably fatal in from a few hours (apparently) to several weeks or several months. The average duration of the general or typhoid cases is about four weeks; that of the meningeal cases somewhat less.

Tuberculosis of the Lymphatic System.

Lymph Nodes.—Etiology.—Tuberculosis of the lymph nodes (scrofula) is very common in children, especially prior to the tenth year of age, and in the female sex; but it may occur in both sexes and at all ages. The process may be widespread or circumscribed, especially to the cervical, bronchial, or mesenteric lymph nodes. In the cervical cases the infection occurs by way of the tonsils, carious teeth, and the cutaneous and mucous surfaces of the head and neck and related cavities (nose, mouth, pharynx, larynx, etc.). The bronchial nodes are involved in all cases of pulmonary infection, in adults as well as in children; the infection may be bronchogenic, or, especially in children, it may occur by way of the intestine, the bacilli penetrating the mucosa and traversing without infecting the mesenteric and other abdominal lymph nodes. Some writers maintain that in many of these cases infection occurs during intra-uterine life, and that there is a variable period of latency or quiescence after birth. The mesenteric and retroperitoneal nodes may become infected in consequence of infected food, especially milk and butter; the disorder is especially common in children (tabes mesenterica). In adults mesenteric involvement is commonly secondary to intestinal ulceration.

Pathology.—The lesions consist of tubercle formation, at first gray or miliary tubercles, and later yellow or caseous tubercles; or of diffuse productive inflammation. The diseased lymph nodes vary in size from that of a pea to that of a walnut or thereabout. Usually one node after another in a chain becomes diseased. At first they are firm and flesh-like, but generally, especially in the neck, they undergo caseation, softening, and may discharge externally. Like changes may occur in other situations. On the other hand, there is some considerable tendency to spontaneous healing, although it may be long delayed; but fibrosis and calcification often eventually ensue, especially in the bronchial and mesenteric nodes. In many of these, notably the bronchial nodes, the tuberculous process is merely latent or quiescent, and at a favorable opportunity may serve as the source of a more or less widespread infection.

Symptoms.—On account of their comparatively superficial position, tuberculosis of the cervical lymph nodes is readily recognized. Node after node, as a rule, gradually increases in size, until the whole chain may be involved. Usually there is little, if any, pain, especially in the early stages; tenderness, as a rule, is slight, but from time to time an acute exacerbation of the condition may supervene, and lead to increased tenderness, some pain, and perhaps redness of the overlying skin: this is likely to be associated with an acute infection of the upper respiratory tract (coryza, laryngitis, bronchitis). The enlarged lymph nodes manifest a marked tendency toward fusion of adjacent nodes, caseation and softening, adhesion with the overlying skin, and the eventual discharge externally of the caseous material; sinuses may persist for a long time, and may ultimately heal with more or less disfiguring scars. In some cases, however, the nodes having reached the size of a bean or cherry, gradually decrease in size and become firm and hard; the process becomes quiescent, arrested, or healed. Tuberculosis of the bronchial nodes may give rise to no symptoms; it is an extraordinarily common, almost constant, condition at the necropsy. Sometimes, however, sufficient enlargement occurs to cause cough (which may simulate that of pertussis), dyspnœa, secondary anemia, and more or less impairment of the general health. The superficial veins of the chest, neck, and head may become distended from interference with the venous circulation in the thorax; there may be relative dulness over and to both sides of the fifth and sixth vertebræ (de la Camp's sign) and perhaps tenderness in the interscapular region (Petruschky's spinalgia); unilateral restriction of the respiratory excursions (Grancher's sign) may be present; and in some cases if the head is strongly flexed backward a murmur may become audible over the sternum or in the suprasternal fossa (Eustace Smith's sign), supposed to result from compression of the aorta by the enlarged lymph nodes. The axillary and inguinal nodes present changes analogous to those of the cervical. In localized tuberculosis of the lymph nodes, constitutional disturbances are commonly slight, and consist of occasional slight fever, increased pulse-rate, and perhaps some loss of flesh and strength; in the event of softening, secondary infection, and

generalization of the process, these general manifestations are likely to become marked. In involvement of the mesenteric nodes (tabes mesenterica), loss of flesh and strength, secondary anemia, and debility usually become aggravated; the abdomen commonly becomes distended; there may be considerable diarrhœa; and the enlarged nodes may be palpable through the abdominal wall. In a few cases, tuberculosis of the peritoneum becomes associated.

Diagnosis.—Tuberculosis must be distinguished from other forms of enlarged lymph nodes, especially syphilis, leukemia, benign and malignant lymphoma, and Hodgkin's disease.

Prognosis.—Tuberculosis of the lymph nodes is a chronic disorder, with a general tendency toward spontaneous recovery—which, however, may be delayed by softening and suppuration. Death may ensue if the process becomes generalized or the general health seriously impaired. The more or less quiescent processes common in the bronchial nodes often, when the subject loses his resisting power, are the source whence pulmonary and more widespread tuberculosis takes its origin.

Treatment.—In addition to the general measures to be presently mentioned, enlarged, but especially enlarging and softened, lymph nodes should be removed surgically, care being exercised not to handle roughly or rupture the softened nodes and thus favor dissemination of the process or its local implantation in tissues unaffected.

Spleen.—Infection of the spleen is usually hematogenic. The common lesion is the miliary or the caseous tubercle, although, especially in children, larger caseous masses may develop; these may become calcified. The organ becomes only slightly enlarged. In chronic suppurating tuberculosis (secondary infections), especially of the bones, amyloid disease of the spleen is likely to develop and give rise to notable enlargement of the organ, which may be readily detected upon physical examination.

Bone Marrow.—Miliary tuberculosis of the bone marrow occurs in practically all cases of generalized miliary tuberculosis, and in some cases in which otherwise only the lungs appear to be involved. The infection is hematogenic. In advanced tuberculosis of other organs, the bone marrow is frequently soft and gelatinous. The bone marrow may also become involved by extension of tuberculosis of the epiphysis or other parts of the bone, whereupon caseous masses are likely to develop.

Serous Membranes.—Pleura.—Tuberculosis of the pleura rarely perhaps may be primary, though this is doubtful; usually it is secondary to disease of the lungs or bronchial lymph nodes, or it may follow disease of the vertebræ, pericardium, peritoneum, etc. It may occur in an acute or a chronic form. The acute cases are represented by the majority of the cases of ordinary serofibrinous pleuritis (which upon good evidence are properly viewed as tuberculous, although this may be demonstrable only upon finding tubercle bacilli in the exudate, by cultural experiments or animal inoculations, or by demonstrating lymphocytosis of the exudate); in other cases, miliary tubercles may be present—usually associated with generalized miliary tuberculosis. In the chronic cases, the

pleura may be greatly thickened with fibrous or fibrocaseous tissue; or dense adhesions may bind together the two serous surfaces so that the pleural sac is obliterated; or rarely large caseous masses, analogous to the "pearl disease" of cattle, may develop. Rupture into the pleural sac of a tuberculous cavity and the establishment of a communication between the respiratory tract and the pleura is the commonest mode of development of pneumothorax or pyopneumothorax. These conditions are discussed in detail under diseases of the respiratory system.

Pericardium.—Tuberculosis of the pericardium is less common than that of other serous membranes; but it is more common than tuberculosis of the myocardium or endocardium. The infection is usually lymphogenic—from the bronchial lymph nodes; but it may be due to extension from the pleura or the mediastinal or other neighboring tissues; and it is rarely the result of hemogenic infection. The process may appear as: (1) The ordinary serofibrinous pericarditis, which like the similar process in the pleura is usually tuberculous; (2) miliary tubercles—due to hemogenic infection; (3) large caseous masses—commonly lymphogenic infection; and (4) dense fibrous or fibrocaseous adhesions binding together the two serous surfaces and more or less completely obliterating the pericardial sac. These conditions are discussed in more detail under diseases of the pericardium.

Peritoneum.—Cases of primary tuberculosis of the peritoneum have been reported, but it is exceedingly doubtful if this ever occurs. The process is usually secondary to tuberculosis of the pleura, pericardium, mesenteric nodes, intestines, genitalia, especially in the female (the Fallopian tubes, etc.); and it may be part of a generalized miliary tuberculosis. The infection may occur by direct extension, by continuity or contiguity of tissue; or it may be lymphogenic or hemogenic. The lesions may consist of miliary tubercles, of larger caseous masses, or of adhesions.

In the miliary type the tubercles are found disseminated more or less generally over the peritoneum; they are commonly most numerous over the intestines, but they may be seen over the spleen, liver, stomach, pelvic organs, and parietal peritoneum. Those miliary tubercles, which are found upon the serous surface of intestinal ulcers and not otherwise in the peritoneum, are commonly not considered as manifestations of tuberculous peritonitis. A large serous exudate is usually the characteristic feature; in some cases the fluid is hemorrhagic in character. The caseous or ulcerative type is a chronic condition, and may be the result of any of the sources of infection except the blood channels. In this type large cheesy masses develop, a plastic exudate is thrown out and gives rise to more or less adhesion of the abdominal organs. The omentum shares in the condition, and large caseous masses may develop therein. The adhesive type is even more chronic, and is characterized by the development of dense fibrous adhesions between the various organs. There is sometimes an absence of exudate, whence the condition is spoken of as dry peritonitis; often there are sacculations with more or less fluid.

Symptoms.—Miliary tuberculosis of the peritoneum presents the usual symptoms of acute generalized miliary tuberculosis, such as fever, increased pulse and respiratory rate, headache, loss of appetite, and progressive emaciation. Certain localizing symptoms, however, may be observed, such as abdominal pain and tenderness, distention, tympanites, and ascites. The bowels may be constipated or loose. The onset of symptoms is usually gradual, though in some cases it is abrupt. The course is commonly not prolonged beyond one or two months. In chronic caseous peritonitis the onset is much more gradual. The patient slowly fails in health, emaciates, and develops moderate fever. After a time abdominal pain is complained of and tenderness and distention are noted. Nausea and vomiting usually appear late in the disease. Owing to the wasting of the abdominal tissues and to the obstruction offered to the circulation in the intra-abdominal vessels, the superficial abdominal veins become prominent. Inflammatory changes about the umbilicus with a discharge of purulent or fecal matter may occur. Palpation reveals a doughiness of the abdomen, marked here and there by rather firm tumor-like masses, which have been found to be made up of intestinal coils and omentum, sometimes enclosing sacculated fluid. The course of the disease may be prolonged over many months and may develop into the adhesive type. In the caseous type there are usually symptoms referable to infection of other parts of the body, as, for example, the lungs and intestines. In many instances the adhesive type is secondary to the miliary or caseous, but some cases are doubtless primary in character. The symptoms are much like those just mentioned, except that there may be no evidences of abdominal distention and ascites; on the contrary, the abdominal walls are likely to be retracted. Pain and tenderness are moderate, and constipation is usually marked. Tuberculosis of the peritoneum is not infrequently associated with hepatic cirrhosis, which is usually the primary condition.

Diagnosis.—The diagnosis of peritoneal tuberculosis is often difficult. In cases of acute generalized disease, which present abdominal tenderness, distention, and ascites, the diagnosis is suggested, though not infrequently the condition has been mistaken for typhoid fever; in the chronic caseous and fibrous types the symptoms and physical signs may lead to confusion with simple peritonitis. The fever is likely to be more irregular in tuberculosis than in typhoid fever, and it does not show the lysis which is characteristic of the latter affection. Abdominal tenderness is more commonly general in tuberculosis of the peritoneum, while in typhoid fever it is likely to be confined to the cecal region. The redness and induration of the tissues about the umbilicus with the discharge of purulent or fecal material therefrom is diagnostic of tuberculous peritonitis. Palpation and percussion of the abdomen usually reveal masses of omental and intestinal tissues. In many instances it may be very difficult to differentiate the simple from the tuberculous type of peritonitis. Fluid may be withdrawn from the abdomen, but it usually does not show the presence of tubercle bacilli; but may reveal

lymphocytosis. Animal inoculation of this fluid, however, may reveal the character of the disease. The detection of tuberculous foci in other parts of the body may be of great assistance in the diagnosis of the peritoneal condition.

Prognosis.—The prognosis depends largely upon the degree of infection of other parts of the body, as for example, if the intestines and lungs are markedly diseased the outlook is grave. Aside from this, tuberculosis of the peritoneum not infrequently becomes spontaneously arrested, its specific character disappearing and dense fibrous adhesions developing. Cases of miliary tuberculosis of the peritoneum are generally fatal, but those of the caseous and adhesive types may undergo a certain degree of resolution. The more there is of fibrous tissue and the less of caseation the greater is the probability of recovery from the immediate effects of the disease. The development of fibrous adhesions may cause considerable disturbance in the functions of the abdominal organs; and relapses are not uncommon in those cases, which seem to be tending toward recovery. In such an event death not infrequently occurs. Tuberculosis of the peritoneum usually warrants a more favorable prognosis than tuberculosis of the meninges, and oftentimes of the pleura.

Treatment.—In addition to the general measures, to be presently mentioned, operative procedures are often of much service, although they should not be lightly undertaken, since many cases after the lapse of several months tend to spontaneous recovery. Recovery also sometimes follows repeated tappings. Yeo speaks highly of iodoform, $\frac{1}{2}$ grain (0.03 gram) in cod-liver oil, 2 drams (8 c.c.) thrice daily; and inunctions of the abdomen twice daily with a mixture of equal parts of iodoform ointment and cod-liver oil. Cœliotomy with or without peritoneal irrigation with physiological saline solution or mild antiseptic solutions, or the use of iodoform or other dusting powders, is much superior to simple paracentesis. Although the operation is frequently followed by excellent results—cure in from one-half to three-fourths of the cases—its mode of action is not definitely known: the access of sunlight and air, the traumatism of the operative manipulations, the access of fresh blood serum with augmented antitoxic or bactericidal properties permitted by removal of the peritoneal fluid, etc., have been credited with the curative properties.

Tuberculosis of Several Serous Membranes; Multiple Serositis; Polyorrhomenitis.—The pleuræ, pericardium, and peritoneum may be involved concurrently in tuberculosis, the infection developing simultaneously or sequentially in two or more of the serous membranes; and the lesions may be equal or disproportionately more marked in the one than in the others. They may consist of miliary tubercles, of larger caseous masses with more or less fluid exudation, or of fibrous or fibrocaseous thickenings and adhesions with varying amounts of fluid, sometimes very little, sometimes considerable. The condition is discussed in more detail under diseases of the peritoneum.

Tuberculosis of the Circulatory System.

Heart.—Tuberculosis of the myocardium is rare; it is always secondary to a focus elsewhere in the body. Four forms are recognized: (1) Miliary tubercles, (2) large caseous tubercles, (3) diffuse tuberculous infiltration, and (4) generalized fibrosis. The first three types are usually recognized grossly, but the fourth may be evident only upon microscopic examination. The heart muscle may be involved secondarily to the pericardium. The auricular and ventricular walls are involved apparently with equal frequency. The lesions of the first type are ascribed to blood infection, and the second to dissemination through the lymphatics. There are no symptoms by which the disease can be diagnosticated; rapid and irregular heart action and sudden death have been noted in certain cases, but there are no symptoms which may not be referable to simple myocarditis. Tuberculosis of the endocardium may appear as valvular vegetations, miliary tubercles over the mural endocardium, or diffuse sclerosis of the endocardium. The affection is rare, though somewhat more frequent than myocardial tuberculosis. It is always associated with acute generalized miliary tuberculosis, and the infection naturally reaches the parts through the blood stream. Cardiac thrombi have been found to contain tubercle bacilli without endocardial lesions being present. There are no symptoms diagnostic of the disease; if the valves are affected a murmur may be heard, but this may be ascribed to a simple endocarditis.

Bloodvessels.—The bloodvessels are sometimes the seat of tuberculosis. The aorta is rarely involved as a result of extension from a caseous bronchial or mediastinal lymph node; the pulmonary veins may contain tubercles in their walls, especially in pulmonary tuberculosis; and the small arteries or veins or the capillaries throughout the body may reveal miliary tubercles, especially in miliary tuberculosis. Miliary tuberculosis itself is the result of the irruption into a bloodvessel of a tuberculous focus somewhere in the body. In cavity formation in the lung, larger or smaller vessels are not uncommonly relatively unaffected, even after extensive disease of the other pulmonary tissues; but eventually, from primary involvement of the adventitia, the wall becomes weakened, bulges (that is, a small aneurysm develops), and may finally rupture and give rise to a more or less copious hemorrhage.

Tuberculosis of the Respiratory System.

Nose.—Tuberculosis of the nasal mucous membrane is usually secondary to a lesion of the lungs or larynx, but a primary focus may be the result of infection by means of a contaminated handkerchief or finger. The disease may appear in one of two forms, as an ulcer or as a tumor-like mass (tuberculoma). Both are rare and practically of equal frequency. Lupus involving the face may by continuity of struc-

ture invade the nose. Aside from constitutional treatment, such agents may be employed as are capable of destroying the infected tissues, the curette and the galvanocautery.

Larynx.—Primary laryngeal tuberculosis is very rare; many writers doubt its existence. Statistics show that it complicates pulmonary tuberculosis in 25 to 30 per cent. of the cases. It may appear early in the course of the lung disease, but more frequently it is seen at a later period. Infection may occur through the respiratory tract, the blood, or the lymph. If the condition is primary, the infectious material has found its way to the part through the inspired air. In the majority of cases of secondary laryngeal infection, those attending pulmonary disease, the sputum probably serves as the infectious medium. The frequency of involvement of the posterior surface of the larynx suggests the probability of sputum infection, especially in bedridden patients. The blood may be the carrier of infection in cases of generalized miliary tuberculosis, while the lymph may act in a similar capacity when there is tuberculosis of adjacent structures. Any portion of the larynx may become affected. Some statistics show that the vocal cords are more frequently diseased than other parts; others show that the epiglottis is the commonest seat. The disease appears primarily as a miliary tuberculosis of the subepithelial tissues; after a time coalescence of the lesions occurs with more or less erosion of the superficial structures, and ulceration results. These lesions usually have a rough, worm-eaten appearance and are situated rather superficially. On the contrary, tumor masses (tuberculomas) may appear, and project into the lumen of the larynx.

Symptoms.—The chief symptoms are hoarseness, pain, difficulty in swallowing, and cough. Hoarseness is usually the earliest symptom, and gradually progresses to almost complete aphonia, which develops in consequence of œdema or destruction of the vocal cords. Pain, which usually does not appear until late in the disease, may be moderate or severe, spontaneous or produced only upon swallowing. In the majority of cases cough is produced by the pulmonary lesions, although irritation of the laryngeal nerves alone may incite cough.

Diagnosis.—The diagnosis is readily made in most cases by a direct examination of the parts; but syphilitic and malignant ulceration must be excluded. The worm-eaten and superficial character of the ulcers and the pallor of the tissues are characteristic of tuberculosis; in syphilis the destruction of the part is much more rapid, there are evidences of inflammation in the surrounding tissues, and the ulceration, which is usually solitary, is likely to be deep. Malignancy characteristically appears later in life than does tuberculosis and syphilis; the lesions are primarily unilateral in position; and marked œdema and inflammatory changes about the ulcer are usually found.

Prognosis.—The prognosis depends upon the extent of laryngeal and pulmonary involvement. A larger percentage of cures is now effected than formerly, owing to perfected methods of local treatment.

Treatment.—Constitutional as well as local measures are important in the treatment of this condition. Much can be done for the early cases of laryngeal disease, but in those in which extensive destruction of the tissues has occurred nothing but a fatal issue is to be expected. Subjects who experience severe pains upon swallowing die not infrequently of inanition. The most efficient treatment is that of tuberculosis in general (to be presently mentioned) and the judicious administration of such drugs as strychnine, nux vomica, pepsin, creosote, and ammonium chloride, etc. Creosote is of value if mixed infection is present in the lungs (shown by cavitation and free expectoration of purulent material). Ammonium chloride (by its stimulating properties) is of service in order to alleviate the marked dryness of the mucous membrane. The local treatment should consist of the application of lactic acid, in a 20 to 80 per cent. solution, to the ulcers. Methylene blue in 2 to 4 per cent. solution, or the colloidal silver salts in 20 to 30 per cent. solution, may be effective. The curette may also be of service. If the condition is far advanced and the patient suffers to a marked degree, sedative applications may be employed, such as cocaine, boric acid, morphine, iodoform, orthoform lozenges, etc.

Trachea and Bronchi.—Tuberculous ulceration of the trachea and bronchi is not infrequently seen in cases of pulmonary and laryngeal infection. The ulcers are usually small and multiple. The bronchial lesions are more frequently seen in the chronic than in the acute form of pulmonary tuberculosis. The ulcers resemble those of the larynx in being rather superficial and worm-eaten in appearance. Infection in most cases is aërogenous or bronchogenic in character.

Lungs.—Etiology.—Pulmonary tuberculosis is the commonest manifestation of the disease. Two pathways of infection are postulated: the respiratory and the alimentary; but the former is the more generally accepted. The respiratory or aërogenous theory presupposes the aspiration of infectious material into the lower respiratory tract, where it becomes deposited upon the mucous surfaces. Direct penetration of the bacilli to the alveoli is questionable; it seems probable that they find lodgment upon the mucosa of the small bronchi and bronchioles, whence they are carried either by the lymph or by phagocytes through the mucous membrane to the peribronchial lymph spaces in which the primary tubercles develop. From these areas dissemination occurs to other parts of the lungs. The alimentary theory presupposes that the ingestion of infectious material is followed by the penetration of the intestinal walls by the tubercle bacilli and by their convection to the mesenteric nodes; thence they pass to the lungs by way of the thoracic duct, the left subclavian vein, the superior vena cava, the heart, and the pulmonary artery; capillary embolism occurs in the lungs and miliary tubercles develop. The majority of cases of pulmonary tuberculosis is due to respiratory infection, but a certain number may be the result of alimentary infection, especially in children, to whom milk may be a frequent carrier of the bacilli. The most important source of pulmonary

tuberculosis is the inhalation of tubercle bacilli brought about by the inspissation and pulverization of sputum from a tuberculous patient.

Pathology.—Tuberculosis of the lungs may develop in one of four forms: (1) Acute miliary tuberculosis (which has already been discussed); (2) acute pneumonic tuberculosis; (3) chronic ulcerative tuberculosis; and (4) chronic fibroid tuberculosis.

Acute Pneumonic Tuberculosis.—Acute pneumonic tuberculosis occurs in two subtypes—the so-called pneumonic and the bronchopneumonic. The so-called pneumonic form is most common in adults, and usually involves a lobe or an entire lung. In general the appearances are much like those of pneumococcic infection of the lung (croupous pneumonia); but in the early stages there is more fluid exudation that gives rise to a peculiar gelatinous appearance, and there is likely to be less fibrinous exudation and more desquamative changes. Definite tubercle formation may or may not be present; in some cases the entire exudation consists of the ordinary inflammatory products with marked proliferation of the alveolar epithelium, all of which proceeds soon to extensive caseation and softening; in other cases, amidst the widespread caseation tubercles also may be found. The bronchopneumonic form is most common in children and young adults. The lesions consist of reddish, later grayish or yellowish foci of bronchopneumonia, which by fusion may lead to involvement of almost if not quite all of a lobe or an entire lung. There is a marked disposition to caseation, and, if the patient survives long enough, to excavation.

Chronic Ulcerative Tuberculosis.—In advanced cases the lesions are quite diversified, consisting of gray and yellow tubercles, foci of bronchopneumonia, diffuse tuberculous infiltration with caseation, excavations, fibrous changes in the lung and pleura, etc. The disease begins with the development of gray tubercles, most commonly in the apex of the upper lobe (most frequently the right), about an inch to an inch and a half below the extreme limit of the lung; that is, about the distribution of the posterior apical bronchus. This has been attributed to inefficient expansion of this region owing to the proximity of the clavicle and to a relatively poor blood supply. The process may also begin in the apex of the lower lobe (about an inch below its uppermost limit, corresponding to the fifth thoracic vertebral spine). Wherever the tubercles first develop, they tend to progress downward; but in some cases the primary lesions are low down, at the base of one or the other lung. When advanced there may be few or many tubercles—of both the gray and the yellow variety; and they may be confined to a small section of one lobe, or they may be scattered throughout an entire lung or both lungs. In cases of rather rapid course, about a more or less obviously primary focus there may be many young tubercles, with comparatively little caseation: the patient has been overwhelmed by the toxemia. In more chronic cases conglomerate tubercles form, and eventually undergoing caseation and softening and leading to ulceration of the wall of a bronchus, discharge their softened contents externally; a so-called cavity results.

These cavities are most common in or near the apex (the seat of the oldest lesions); they vary much in size and in number, and they are usually surrounded by more or less fibrosis, which surrounds also the adjacent bronchi. These destructive changes are commonly due to the association of pyogenic cocci with the tubercle bacilli; that is, they are evidences of mixed infection. In some cases there is more or less fibrosis aside from that about cavities; this fibrosis may partly or completely enclose a tuberculous focus, isolating it from the remainder of the lung; in other cases strands of fibrous tissue pervade and may ultimately replace the tuberculous focus—the process has become healed.

Chronic Fibroid Tuberculosis.—In addition to the aforementioned fibrosis about a tuberculous focus, more or less extensive fibrosis may occur concurrently with an active tuberculosis—of the chronic ulcerative or chronic bronchopneumonic type; or it may occur in association with chronic pleural adhesions. The fibrosis may be so extensive as almost if not quite completely to replace the pulmonary tissue of an entire lobe or more; there may be cavity formation; and there is usually dilatation of the smaller and medium-sized bronchi (bronchiectasis). In all cases of tuberculosis of the lung, of whatever variety, pleural adhesions are the rule; empyema is quite common; and from rupture of a tuberculous cavity into the pleura pyopneumothorax may develop.

Symptoms.—Acute Pneumonic Tuberculosis.—In the adult the onset is often quite like that of ordinary croupous pneumonia; the physical signs are those of solidification of a lobe or an entire lung; and the course of the disorder is not notably different from that commonly seen in pneumococcic infection of the lung. Suspicion of the real nature of the disorder commonly is not entertained until the expected crisis fails to occur. The tuberculous nature of the infection, however, is sometimes suggested by antecedent ill health of the patient, an ill-explained cough lasting for several months, a more or less well-defined tuberculous lesion in the lung, bloody rather than rusty sputum, sweating, emaciation, remittent rather than continuous fever, slight leukocytosis, and later the detection of tubercle bacilli in the sputum. The bronchopneumonic type sometimes develops in a person previously well, but it is most likely to occur in subjects in general ill health, or in children after measles, pertussis, and other infections. The symptoms and physical signs, in the beginning at least, are quite like those of bronchopneumonia developing under other circumstances and due to other microörganisms; but at the end of ten days or two weeks the patient, especially if a child, seems to fail markedly in general health, to become emaciated, to develop chills, sweats, and hectic fever, and, if he does not die soon, gradually presents the common manifestations of chronic ulcerative tuberculosis.

Chronic Ulcerative Tuberculosis.—This, the common type of tuberculosis of the lungs, may reveal itself at the onset in a variety of ways. The first symptoms may be those of general ill health, with loss of appetite and of weight, without localizing symptoms; or those of anemia, especially common in young girls, and usually mistaken for

chlorosis; or those of bronchitis (a "cold" that does not get well), laryngitis, pleuritis, hemoptysis, fistula in ano, etc.

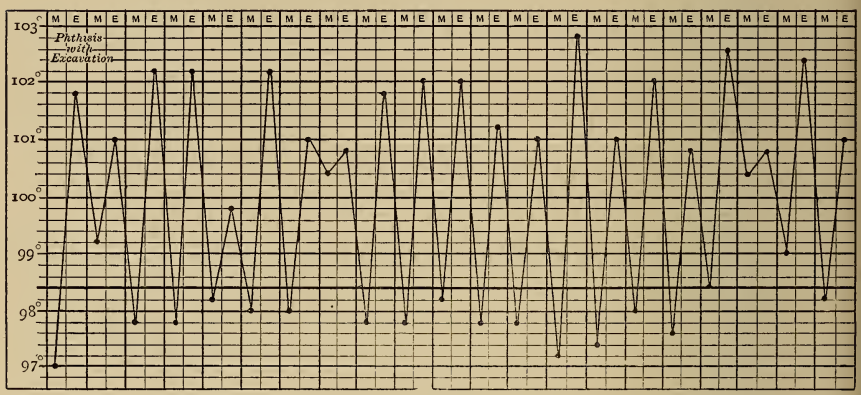
The classical symptom of pulmonary tuberculosis is cough, although in a few cases it is absent until an advanced stage of the disease. In many cases it is the first symptom that calls attention to the pulmonary condition. It may be severe and very harassing to the patient, but, on the contrary, it is often mild and disturbs the patient only upon arising in the morning. In the beginning the cough is dry, but as bronchial irritation increases and disintegration of the pulmonary tissues develops, sputum appears. At first this is mucoid in character, but when secondary infection by pyococci takes place it becomes mucopurulent. In an advanced stage, the sputum often contains small, hard, coin-like masses, by which it is designated as "nummular" in character. Elastic tissue evidencing the destruction of the lung is often found, not only in these masses, but also free in the sputum. Except in the earliest stage tubercle bacilli also are found; to these late in the disease other microörganisms are added, chiefly staphylococci, streptococci, pneumococci, and Micrococcus tetragenus. Early in the disease the sputum usually contains more or less bronchial and alveolar epithelium, the latter having been held at one time to be diagnostic of the disease. When a mixed infection is present pus cells are found.

Hemoptysis not infrequently is the first manifestation. Usually only a moderate amount of blood is lost, and this is ascribed to pulmonary congestion. In some cases small, frequently repeated hemoptyses occur during the course of the disease, and for several days following such manifestations the sputum is blood-streaked. Later in such cases larger hemoptyses may ensue, but they rarely lead directly to death. In the latter stages the blood is derived from eroded bloodvessels adjacent to or within cavities. There is a surprisingly large number of cases in which no hemorrhage occurs at any time, not even when large cavities are present.

Loss of weight is an important early sign, and in many instances precedes the development of cough, expectoration, and hemoptysis. If it be associated with a slight rise of temperature in the afternoon or early evening, suspicions should be immediately aroused as to its tuberculous basis. The association of these two symptoms is of vital importance in the diagnosis of the disease. In the very earliest stage the temperature may be subnormal; but usually when the patient comes under observation, there is a little fever, amounting usually to 1° or 1.5° F., and the temperature is likely to be very unstable. As the disease advances the temperature increases, so that finally it becomes typically hectic in character, the morning temperature being subnormal and the afternoon temperature (3 to 6 P.M. or thereabout) reaching 103° to 104° F. (Fig. 3). The highest temperatures are seen in acute pulmonary involvement; the development of an acute pneumonic process in the chronic form of tuberculosis adds materially to the temperature. The fever is referable to the toxemia. The pulse rate is also increased,

even in an early stage; later it is usually increased proportionately with the temperature, and it is characteristically of low tension and of small volume. The respiratory rate is increased, and dyspnœa may be noted early in the disease. As the disease advances, coincidently with the development of cavities, the dyspnœa may become marked. This, however, is not always dependent upon the amount of lung involvement, since in early cases there may be marked dyspnœa, due doubtless to a toxic influence on the respiratory centre. On the contrary, in some advanced cases there is remarkably little dyspnœa.

Fig. 3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not present this symptom until the disease is far advanced. Vomiting is a distressing symptom manifested in some cases and has no connection apparently with gastric disease, but rather with the cough. It is seen more frequently in the morning than at any other time of the day, and is induced by severe attacks of coughing which not infrequently follow breakfast. It usually attends cavity formation. There is no constant symptom referable to the intestinal tract, though the majority of patients are constipated. If a persistent diarrhœa is present, tuberculosis of the intestines may be suspected. Often the stools give evidence of deficient biliary secretion. From time to time the patient complains of abdominal pains, which are often referable to constipation and biliousness. As the disease advances some subjects show a decreased elimination of urine owing to nephritis, which is likely to develop sooner or later. Hoarseness not infrequently appears in the bedridden patient in consequence of a terminal laryngeal infection; in a few cases the distressing symptoms of laryngitis develop at an earlier period. From time to time, even from the earliest stage of the disease, headache is complained of, and is due doubtless to the toxic condition of the brain centres or to defective excretion, or both. Coincidently with the development of these symptoms the patient gradually loses in strength and weight; in the very chronic cases extreme emaciation is seen, and is associated with a hectic flush of the cheeks, hollow, staring eyes, etc.

In those patients in whom tuberculosis has assumed a very chronic form incurvation of the nails and clubbing of the fingers and sometimes of the toes develop. These require considerable time for their development, and are probably dependent upon a toxic influence and peripheral passive congestion. Such extremities are always cyanosed and cold, as a result of defective circulation.

Physical Signs.—In advanced cases the disease may be disclosed by a long, narrow, and flat chest, with an acute epigastric angle, and protruding scapulæ (alar or winged chest). In many cases, however, these signs are absent for a long time. The earliest physical signs consist of deficient expansion, lagging behind or depression, of one apex (usually the right) anteriorly or posteriorly; increased tactile and vocal fremitus; impaired percussion resonance (sometimes for a time masked by compensatory inflation or emphysema of the surrounding portion of the lung, which then yields a vesiculotympanitic note); and weak or harsh, vesicular breath sounds with prolongation of the expiration. In the early stages it is often difficult to determine whether these signs are more pronounced that the normal differences between the two apices. Soon, however, fine crackling and perhaps fine bubbling rales become apparent; and the breath sounds may take on the so-called cogwheel character (which occurs also in other conditions). As the disease advances (the infiltration of the lung increases) these signs become more marked and extend over a wider area, and very commonly they develop at the other apex, or in the apex of one of the lower lobes. The percussion note becomes actually dull, and tactile and vocal fremitus much exag-

gerated, the breath sounds bronchovesicular or bronchial, the rales much more numerous, and to those previously present coarser bubbling (bronchitic) rales are added. Pleural frictions also are frequently audible, but it is often almost impossible to distinguish them from rales, especially when rales are numerous. Over an area of excavation (cavity formation) the tactile and vocal fremitus may be much increased unless the overlying pleura is much thickened; bronchophony and pectoriloquy are likely to be elicited; the percussion note is vesiculotympanitic or distinctly tympanitic, and the so-called cracked-pot sound may be elicited, if the cavity is near the surface, is of fair size (about two inches or more in diameter), has thin resilient walls, and communicates with an open bronchus. The occurrence of tympany over a previously dull area following the evacuation of considerable sputum is quite significant of a cavity, but other changes in the percussion note may occur, although they are inconstant and of minor diagnostic importance: such as, variations in the note, depending upon whether the patient is in the upright or the recumbent position (Gerhardt's sign), which occurs over cavities of unequal axes, partly filled with fluid, and communicating with an open bronchus; a louder and higher pitched tympanitic note when the patient's mouth is open as contrasted with the note elicited when the mouth is closed (Wintrich's sign); inspiratory increase and expiratory decrease in the loudness and pitch of the note (Friedreich's sign); the breath sounds become bronchial, cavernous, or amphoric in character; and coarse bubbling or gurgling, sometimes resonant, rales may be heard. Myotatic irritability (myoidœma) is quite common in advanced cases. Over the diseased area the intercostal muscles may be rigid; and there may be local tenderness, due sometimes to localized pleuritis, sometimes perhaps to toxic influences on the nerves. Percussion over an area of excavation, especially in the subclavicular region, may lead to immediate cough and expectoration (Erni's tapotage sign). A systolic murmur is often audible over the subclavian artery, and is referable to pleural adhesions, compressing or distorting the artery. Early in the course of the disease the heart usually reveals no abnormality, aside from accentuation of the pulmonary second sound, the result of congestion of the lungs; later a functional systolic murmur commonly develops, and a systolic murmur of relative mitral insufficiency—from dilatation; and the area of cardiac dulness may become increased—which, however, is due to retraction of the borders of the lungs (especially the left) rather than to enlargement of the heart. Cardiopulmonary murmurs also are common. The liver and perhaps the spleen may be enlarged—the liver from fatty infiltration; both organs from tuberculosis or amyloid disease.

Chronic Fibroid Tuberculosis.—The chief symptoms consist of cough, mucopurulent expectoration, and dyspnœa; the sputum may be fetid in the event of bronchiectasis—which is common. Recurring attacks of hemoptysis are rather frequent: I have seen a subject in whom these occurred over a period of more than twenty years, with maintenance of good general health, the weight being about or nearly two hundred

pounds. From time to time there may be attacks of more or less fever. The physical signs are those of chronic interstitial pneumonitis: distortion and insinking of the chest, with uncovering and perhaps displacement of the heart; and over the affected area dulness on percussion, increased tactile and vocal fremitus (except in the event of pleural adhesions which are rather common), bronchovesicular or bronchial breath sounds, and fine or coarse bubbling rales.

Diagnosis.—*Acute pneumonic tuberculosis*, since it resembles ordinary croupous pneumonia in its mode of onset and its course, is commonly not recognized until the expected crisis does not occur; but certain of the aberrant phenomena already mentioned may excite suspicion of the tuberculous nature of the infection and sometimes lead the observing physician to an early diagnosis. *Chronic ulcerative tuberculosis* is disclosed by any of the aforementioned early symptoms, especially slight or progressive loss of weight; early subnormal temperature, but rather soon slight afternoon fever; rapid and unstable pulse; cough with or without slight expectoration; even slight hemoptysis; and the physical signs of beginning infiltration of the lung—usually one apex. One should endeavor to make the diagnosis before tubercle bacilli appear in the sputum; their detection as well as the presence of elastic fibers makes the diagnosis conclusive. In the event of doubt recourse may be had to one of the several tuberculin tests (page 125). Radiographic examination often enables one to detect a tuberculous focus in the lung otherwise insusceptible of recognition. In the differential diagnosis one must exclude syphilis, actinomycosis, streptothricosis, and malignant disease of the lungs. Syphilis of the lung is rare, and the diagnosis can seldom be made with certainty; the disorder may be suspected upon the development in a known syphilitic subject of the physical signs of chronic interstitial pneumonitis, with perhaps bronchiectasis of the lower lobe, and the absence from the sputum of tubercle bacilli; but one must remember that tuberculosis of the lungs may occur in a syphilitic subject and that tubercle bacilli may not be found for some time; a positive Wassermann reaction may be of some assistance. Actinomycosis and streptothrical infections are recognizable by the detection in the sputum of the specific infectious agent. In malignant disease there is usually little if any fever; the physical signs are those of irregular solidification—in one, sometimes in both, lungs; secondary anemia and cachexia soon supervene; the sputum is often blood-tinged or it may be distinctly bloody, and may reveal tumor cells, but no tubercle bacilli. *Chronic fibroid tuberculosis* is readily recognized by the physical signs of chronic interstitial pneumonitis and the presence of tubercle bacilli in the sputum.

Prognosis.—*Acute pneumonic tuberculosis* usually leads to death in from four to eight weeks; rarely the progress of the disorder becomes somewhat stayed, and the process may become chronic, but a fatal termination seems inevitable. Death in some of the chronic forms of pulmonary tuberculosis may be finally induced by the acute pneumonic or bronchopneumonic process. In *chronic ulcerative tuberculosis* the

prognosis depends upon the extent of the process when detected, the resistance of the individual, his occupation and other social environment, the ability or lack of ability to place himself under the best hygienic, medical, and other conditions, etc. A poor general condition, poverty, inappropriate and insufficient food, exhausting work, mental cares and anxiety, etc., are potent deterrents of recovery. In incipient and even moderately advanced cases, under favorable circumstances and the best hygienic surroundings, the outlook is good, if not for recovery, at least for comparative comfort and prolongation of life. *Chronic fibroid tuberculosis* may be a fortunate outcome of the chronic ulcerative process, but whether developing thus or otherwise, it may last for many (twenty or more) years.

Classification.—The American Sanatorium Association, approving a committee report, suggests the use of the following classification of patients upon admission and discharge and for ultimate results; the classification is equally applicable in private practice:

1. On Admission.—These definitions indicate the furthest extent of the disease and the greatest severity of symptoms that a patient can present and still belong to the stage defined. All patients beyond the incipient stage fall under the moderately advanced stage unless the physical signs and symptoms exceed those of the moderately advanced stage, when they should be classified as far advanced.

Incipient.—Slight or no constitutional symptoms (including particularly gastric or intestinal disturbance or rapid loss of weight); slight or no elevation of temperature or acceleration of pulse at any time during the twenty-four hours. Expectoration usually small in amount or absent; tubercle bacilli may be present or absent. Slight infiltration limited to the apex of one or both lungs or a small part of one lung. No tuberculous complications.

Moderately Advanced.—No marked impairment of function, either local or constitutional. Localized consolidation, moderate in extent with little or no evidence of cavity formation; or infiltration more extensive than under incipient. No serious complications.

Far Advanced.—Marked impairment of function, local and constitutional. Marked consolidation of an entire lobe; or disseminated areas of beginning cavity formation; or serious complications.

Miliary Tuberculosis.

2. On Discharge.—*Apparently Cured.*—All constitutional symptoms and expectoration with bacilli absent for a period of three months and the physical signs to be those of a healed lesion.

Arrested.—Absence of all constitutional symptoms; expectoration and bacilli may or may not be present; physical signs stationary or retrogressive; the foregoing conditions to have existed for at least two months (a somewhat arbitrary period, intended to cover the cases, which frequently occur, of patients who leave the sanatorium for various reasons, contrary to advice, after a stay of a few weeks, although all active symptoms may have ceased completely soon after entrance).

Improved.—Constitutional symptoms lessened or entirely absent; physical signs improved or unchanged; cough and expectoration with bacilli usually present.

Unimproved or Progressive.—All essential symptoms and signs unabated or increased.

Died.

3. Ultimate Results.—*Cured.*—All constitutional symptoms and expectoration with bacilli absent for a period of two years under ordinary conditions of life.

Well.—Patients who fulfil all the conditions required under "cured," but about whose sputum no definite information can be obtained.

Arrested, Improved, Progressive.—As above.

Dead.

Tuberculosis of the Digestive System.

Mouth.—Tuberculous ulceration of the mouth and tongue is rare, and is usually secondary to laryngeal or pulmonary involvement. The affection simulates syphilitic and malignant ulceration. The tuberculous lesion is commonly solitary, sharply outlined, and pale in color, with an area of œdematous tissue often surrounding it. It is superficially located, at least in its early stage. The syphilitic lesion is generally an ulcerated gumma, and is deeply placed. The malignant (carcinomatous) ulcer occurs usually later in life, and considerable induration usually appears about its margin. A tuberculin test may serve to dispel any doubt in regard to the diagnosis, for the ulcerated tissue is likely to show some reaction if tuberculosis is present.

Tonsils and Pharynx.—Tuberculosis of the tonsils may be primary or secondary. It is not improbable that infection finds its way into the system in many cases through these tissues. However, it seems likely that the majority of cases are secondary and are induced by sputum from an infected lung. The disease appears as caseous foci adjacent to the crypts, and is very common in the advanced stage of pulmonary tuberculosis. The pharynx is even less frequently the seat of tuberculosis than is the oral mucous membrane. The miliary type is the commonest, and this by progress or confluence of the lesions may give rise to ulceration, which may occur in advanced pulmonary tuberculosis.

Œsophagus.—The œsophagus is rarely the seat of tuberculosis. Occasionally caseous bronchial and tracheal lymph nodes erode through the œsophageal wall and cause ulceration therein. More rarely the lesion develops in consequence of an abrasion of the mucosa; this has been noted in a few cases of malignant ulceration, in which tubercles have been noted in the wall.

Stomach.—Aside from the development of tuberculous masses in the peritoneal tissues of the gastric wall coincidently with tuberculous peritonitis, tuberculosis of the stomach is very rare. The usual manifestation is ulceration of the mucous membrane, which is somewhat more frequently found in infancy and childhood than in later life. The

lesions may be solitary or multiple, commonly the latter, and they are superficially placed. In a few cases tuberculosis has been known to become engrafted upon a previously existing simple ulcer. There are no specific symptoms. It may be associated with intestinal ulceration.

Intestines.—The frequency of involvement of the small intestine increases progressively toward the ileocecal valve, since the lymphoid tissue is the seat of predilection and this tissue is present in abundance in the ileum. The appendix and cecum are also commonly diseased. The remainder of the large intestine is affected more frequently than the duodenum or jejunum. The immunity of the duodenum to infection has been ascribed to the acid intestinal contents and to the rapidity of the intestinal current. The source of intestinal infection is chiefly infected milk and butter and the swallowing of tuberculous sputum. In infancy and childhood the bacilli seem capable of penetrating the walls of the intestines without inducing any lesions, and they are then carried to the mesenteric nodes, where a focus of disease is set up. Such permeability of the intestinal mucosa seems not to obtain in adult life. Intestinal ulceration is noted rather more frequently in the latter period of life. Alimentary infection is not the sole source of the disease, for in cases of acute generalized miliary tuberculosis the bacilli are carried by the blood and give rise to the characteristic tubercles.

Pathology.—The ulcer is the usual form of intestinal tuberculosis. It is probably produced by the coalescence of several adjacent foci and the erosion of the mucous surface. The lesion has a ragged, punched-out appearance, with an elevated and somewhat indurated margin. Congestion of the adjacent tissues may or may not be present. Typically the ulcer extends along the gut in a circular manner; this has been ascribed to the infection following the course of the lymphatics. The ulcers vary in size from that of a millet seed to a diameter of four or five inches. On the serous surface of the bowel opposite the lesions miliary tubercles are commonly found. It is rare to find that an ulcer has perforated, such an accident being apparently inhibited by an overgrowth of the tissues at its base. When perforation occurs adhesions develop between the adjacent coils of intestines, indicating a localized peritonitis; at times a generalized peritonitis is the consequence. An unusual form of intestinal tuberculosis is that in which marked hyperplastic changes take place without notable caseation and ulceration. This may be found in any part of the intestine, but there is a predilection for the cecal tissues; a certain degree of stenosis may co-exist. A rectal or anal fistula may be the result of infection of the lowermost portion of the intestinal tract. In cases of pulmonary tuberculosis 4 to 5 per cent. show such fistulæ, which may be clinically the primary lesion.

Symptoms.—The symptoms are indefinite; some patients have diarrhœa, while others exhibit no symptoms referable to the bowels. Diarrhœa with abdominal pains and tenderness in a tuberculous patient is always suggestive of intestinal involvement. Involvement of the colon seems to be provocative of diarrhœa more frequently than involvement

of the small intestine only. Intestinal infection complicates pulmonary tuberculosis in from 60 to 75 per cent. of the cases, especially in adults.

Diagnosis.—It is practically impossible to differentiate intestinal tuberculosis from simple enteritis or enterocolitis. Bloody stools are suggestive of ulceration. If the diarrhœa is refractory to medication, the condition is likely to be tuberculous in character. The finding of tubercle bacilli in the feces is not necessarily diagnostic. In children the diagnosis is made with even greater difficulty than in adults. If a fistula is present the feces may show traces of pus.

Prognosis.—The prognosis is unfavorable, although in some cases cicatrization and healing of the ulcers ensues. Doubtless the condition hastens a fatal issue, on account of the deterioration of the intestinal functions and the increased toxemia promoted by a large ulcerated and infected surface.

Liver.—Tuberculosis of the liver develops mainly as a result of infection through the hepatic artery and the portal vein; it is possible that some cases are referable to the entrance of infectious material through the lymphatics from the gastrohepatic nodes and through the biliary channels. The commonest sources of infection are the lungs and the intestines, coincidently with the development of acute generalized miliary tuberculosis. The commonest type of hepatic involvement is the miliary, in which the characteristic tubercles are seen, not only in the parenchyma of the organ, but frequently upon its peritoneal surface. The organ is likely to be moderately enlarged. Another type is that in which large caseous masses appear; these sometimes soften and give rise to abscesses and cavities. The condition is very rare. A questionable type, tuberculous cirrhosis, has been described in which there are no lesions except hyperplastic changes in the connective tissue. There are no characteristic symptoms, except occasional jaundice, in miliary and caseous tuberculosis of the liver, but upon examination the organ may be found moderately enlarged. In chronic suppurative tuberculosis elsewhere in the body (bones, etc.), amyloid disease may coexist and the organ may be considerably enlarged. In a few of the rare cases of tuberculous abscess the mass protrudes from the costal margin.

Pancreas.—Tuberculosis of the pancreas resembles that of the liver, since the miliary type is the commonest, though the organ is much less frequently involved than the liver. It has been stated that this condition is a pathological curiosity and that involvement of the pancreas is one of the rarest manifestations of tuberculosis. A more thorough study in recent years reveals the fact that the organ is not uncommonly affected, and in children as many as 44 per cent. of cases of generalized disease have been found to show tuberculous pancreatitis. In adult life the condition is much less frequently seen. Cases of primary involvement have been described, but these lack substantial proof. It is probable that every case is secondary to some other focus in the body, and that in a preponderance of these cases infection reaches the organ by way of the

blood channels, that is, coincidently with a generalized miliary disease. There are two other possible methods of entrance of infection—by the lymphatics and by the pancreatic ducts. A few cases have been reported in which large caseous foci were found in the parenchyma of the organ, and in which there was associated involvement of the adjacent lymph nodes. It is likely that in some of these instances the true condition was enlargement and caseation of the nodes, with encroachment upon but no direct involvement of the pancreas. No authentic cases have been described in which infection took place through the ducts from the intestines. There are no symptoms indicative of pancreatic tuberculosis.

Tuberculosis of the Genito-urinary System.

Male Genitalia.—Tuberculosis of the testicle is not uncommon between the ages of twenty and forty years. In the majority of cases, the process is secondary to a focus elsewhere (hemogenic infection), or it follows tuberculosis of some other portion of the genito-urinary system (infection occurring along the vas deferens). The process is usually unilateral, though it may be bilateral. It develops in an insidious manner in the epididymis as a small firm mass; later the body of the testicle may become invaded, and the lesions undergoing caseation and softening may form a connection with the overlying skin and discharge externally, leaving sinuses; or the process may extend along the vas deferens, infecting it, and later involving the seminal vesicles, the prostate, the bladder, the ureters, the kidneys, etc. The semen being infected may lead to tuberculosis of the female genitalia.

Female Genitalia.—The Fallopian tube is more frequently tuberculous than any other part of the female generative system. It has been stated that from 8 to 16 per cent. of specimens removed at operation for inflammatory disease are found to be tuberculous. The source of infection may be the blood, the peritoneum, or the lower genital tract. The disease is primarily one of tubercle formation within the mucosa; this eventually gives rise to nodular, caseous masses and general destruction of the tubal and oftentimes of the ovarian tissues. In many cases of tuberculous peritonitis the tubes are the nidus from which infection is disseminated. There are no specific symptoms aside from those common to ordinary tuboövarian disease. The uterus may present evidences of tuberculosis primarily, as miliary tubercles in the endometrium; after a time large cheesy masses may appear. Infection may occur from the tubes, vagina, or from the circulating blood; tuberculous semen may be responsible for some cases. The disease has been diagnosticated by curettage and the removal of particles of the endometrium in which tubercles and bacilli have been found.

The *mammary glands* are rarely the seat of tuberculosis. The sources of infection are adjacent lymphatic and osseous lesions. Primarily the condition is one of miliary tuberculosis, followed by the production

of caseous foci, from which sinuses develop to the cutaneous surface. Multiple areas of ulceration are not infrequently seen. The diagnosis is confirmed by histological examination of tissues or by finding tubercle bacilli in the purulent material.

Urinary System.—Renal tuberculosis is present in from 40 to 50 per cent. of cases of pulmonary infection. Recent studies have shown that the condition is much commoner than was formerly considered. The infection may be hemogenic, lymphogenic, or urogenic. The commonest type is miliary tuberculosis, which is induced by blood infection, as it is in all other organs of the body. The lesions are bilateral; the cortex especially shows the presence of the characteristic tubercles, associated with which there is usually moderate hyperplasia of the interstitial tissues. Disease of the retroperitoneal nodes may extend to the kidney (lymphogenic infection), in which event the condition is generally unilateral. One rather large caseous focus may mark the disease. These two types have no special symptomatology; the detection of tubercle bacilli in the urine, though of great importance, may not necessarily signify renal tuberculosis, since it has been stated that tubercle bacilli may be excreted by tuberculous subjects in whom the kidneys appear to be unaffected. Some of the supposed tubercle bacilli, however, are doubtless smegma or other acid-fast organisms. The kidneys may become diseased as the result of an infection from the bladder through the ureters (urogenous infection), the ureters often showing no evidences of tuberculosis. Commonly the infection passes first up one ureter and then after a variable period up the other. Gonorrhœa has been suggested as a predisposing cause. The renal pelvis and calyces are first affected; thence the medullary and cortical parts are attacked. Often there is considerable dilatation of the pelvis, in consequence of more or less stenosis of the ureter. The condition is slowly progressive, caseation and widespread destruction of the tissues occurring, so that usually the kidney after a time assumes a multiloculated appearance. The walls of the cavities are rough, ragged, and necrotic, and it is not unusual to find the parenchymatous tissue completely destroyed, and the organ considerably enlarged (pyonephrosis). Hematuria, pyuria, and bacilluria are symptoms. Upon examination the enlarged organ or organs may be well outlined, and a certain amount of tenderness may be elicited over the lumbar region. The condition is to be differentiated from malignant disease of the kidney. The bladder may be the seat of tuberculosis; infection may occur through the urethra by means of infected instruments or coincidently with gonorrhœa, or by way of the blood stream; or in consequence of kidney involvement; or from the reproductive organs of the male sex. As a consequence the vesical wall becomes invaded by tubercles, caseous masses, or it becomes ulcerated. The symptoms are those of cystitis, together with pyuria and bacilluria. The urethra is rarely the seat of tuberculosis. The predisposing factors are gonorrhœa, stricture, and phimosis. Infection may occur from the use of infected instruments or, especially in the female, it may follow tuberculosis of

the lower genital tract; the condition is much less likely due to a descending infection.

Tuberculosis of the Nervous System.

Brain.—Tuberculosis of the cerebral meninges is associated with generalized miliary disease in about 75 per cent. of the cases, and the infection, therefore, is chiefly hemogenic in type. The disorder is most frequently seen in childhood, especially prior to the sixth year. Meningeal infection is not an infrequent complication of tuberculosis of the reproductive and osseous system; moreover, it has been shown that it may be a sequel of operations for the relief of bone and joint disease, and that it is not infrequently a sequel of measles and whooping cough, in which it is probable that the primary lesion is present in the cervical or bronchial lymph nodes. On the contrary, meningeal disease may arise in consequence of lymphatic dissemination, or by direct continuity of structure. Tuberculosis of the eye, ear, and cervical nodes may set up the condition. A few cases have been reported in which the infection occurred through the cribriform plate of the ethmoid bone from a tuberculous rhinitis. Primary cases of meningeal tuberculosis are of doubtful existence.

Pathology.—The disease usually appears as a leptomeningitis, but in some instances it is a true meningo-encephalitis. The base is more commonly affected than the convexity. There are two chief types of the disease: one in which miliary tubercles are found, and the other in which large caseous nodules (so-called solitary tubercles) are encountered. The condition may be localized or diffused throughout the meningeal surfaces. The miliary tubercles may be scattered over the serous membranes; with these there is always more or less exudate present, which serves to flatten the convolutions. The lateral ventricles are usually found to contain an excess of fluid, and this, likewise, may be seen in the third and fourth ventricles, the condition being known as internal hydrocephalus. In the other type one or more large caseous nodules are present, usually over the basal portions of the brain. An excess of fluid in the ventricles usually co-exists. Occasionally chronic leptomeningitis is manifested by thickening of the membranes, and is associated with the presence of a few small caseous masses, the condition being indicative apparently of a healing process. Similar lesions may be found in the spinal cord and its meninges.

Symptoms.—When meningitis supervenes in a case of acute generalized miliary tuberculosis, its attending symptoms overshadow all those previously existing. The onset may be insidious or abrupt, usually the former. There may be a period of vague prodromes during which languor, irritability, headache, vomiting, and constipation may appear. Difficulties in speech are sometimes noted early in the condition. After a time certain characteristic symptoms appear, so that the disease may be divided into three stages: (1) the stage of irritation which is referable

to the meninges and cortex; (2) the stage of pressure, referable to the effusion into the ventricles; (3) the stage of paralysis, referable to increased pressure and extension of the disease to the medulla oblongata. The symptoms of the first and second stages usually overlap. In the early stage the face is commonly flushed, and by stroking the skin upon any part of the body the *tache cérébrale* is usually obtained. The pupils are dilated and photophobia may be marked, especially in adults. Headache is a common symptom, though more frequently noted in adults than in children, and it is likely to be frontal in the former and generalized in the latter. Pains may be complained of in various parts of the body, as in the abdomen, spine, knees, etc., and these joint exacerbations may be so severe that acute rheumatism may be simulated. The decubitus is characteristic, the patient lying upon one side with the legs drawn up, and he resents any disturbance. Incessant vomiting may be present; this is of the cerebral type, sudden, projectile in character, unattended by nausea, and occurs independently of taking food. Constipation is a common symptom usually relieved by aperients, but in some instances there seems to be almost complete paralysis of the bowel. Diarrhœa may be a symptom; this is likely to be present if intestinal tuberculosis is a complication. The temperature is usually not very high, but irregular in the early stage, while in the paralytic stage it may reach as high as 108° F. The pulse is likely to be slow and irregular in the early stage, but later it becomes quickened. In the early stage deep, slow respirations are observed, to be followed later by those of the Cheyne-Stokes type. Retraction of the head and stiffness of the neck are usually not marked, and in many instances they are absent. To a great extent these symptoms depend upon involvement of the posterior basal portions of the brain. Convulsions may appear in any one or all of the stages of tuberculous meningitis; those of the second stage are most distinctive, inasmuch as they may serve to determine the locality of the inflammation. Paralysis, coma, and death may terminate the condition. The symptoms of the solitary tubercle are those of brain tumor.

Diagnosis.—Among the diseases which may simulate tuberculous meningitis are typhoid fever, pneumonia, brain tumor, brain abscess, and thrombosis of the cerebral sinuses. The probability of confusion is greatest in the early stage of the disease, for as it progresses distinctive symptoms appear. The temperature is typically lower in meningitis than in typhoid fever, and the irritability is greater. In the former, the abdomen is retracted, while in the latter it is distended and tympanitic. A rather rigid position and a lateral decubitus are seen in meningitis, and a relaxed position and a dorsal decubitus are common to typhoid fever. The Widal reaction and the results of a study of fluid removed by lumbar puncture should eliminate any doubt. The temperature, decubitus, and abdominal condition in pneumonia more closely resemble typhoid fever than meningitis. In brain tumor the condition is prolonged and the gradual paralysis is very different from that of meningitis;

optic neuritis is likely to be much more marked than in meningitis. Exacerbations of temperature and rigors are characteristic of cerebral abscess; these are absent in tuberculous meningitis, at least in its early stage. Localized venous engorgement about the eyes and temples and the presence of hemorrhage in the fundus of the eye may serve in some cases to differentiate thrombosis of the cerebral sinuses from meningeal tuberculosis.

Prognosis.—The prognosis depends somewhat upon the extent of the inflammation, since if it is localized, recovery may possibly occur, but as the condition is usually generalized, the disease is looked upon as fatal. The course of the average fatal case is from three to four weeks. When the disease is generalized over the convexity, the duration is usually much shorter than when it has the basal distribution.

Tuberculosis of the Bones and Joints.

Tuberculosis of the bones and joints occurs in various forms, such as caries of bone, "white swelling," etc. One-half of the cases develop prior to the twentieth year; the sexes are affected with equal frequency. Traumatism seems to have some predisposing influence; though the bacteria do not necessarily enter the system through the channels thus induced, the vitality of the part is diminished by reason of the injury. Infection gains entrance to the part from a previously existing lesion in the majority of instances by the blood stream. In some cases the lymph stream cannot be eliminated from consideration. The primary focus may be situated in the lungs, genitalia, or lymph nodes. Though primary cases of bone and joint tuberculosis have been described, it is likely that, at least in the majority of the cases, a latent focus of disease was present, probably in a lymph node, as for example in the cervical or bronchial groups. So-called primary cases have been observed to follow infections such as measles, whooping cough, scarlet fever, etc. According to Cornet the disease attacks the bones and joints in the following order of frequency: the spinal column, the hip-joint, the small joints of the hands and feet, the knee-joint, the long bones, the ankle-joint, the tarsus, and the elbow-, shoulder-, and wrist-joints. The sternum, ribs, cranial bones, and pelvis are rarely tuberculous.

Pathology.—Of the bony structures, the cancellous or spongy parts are the seats of predilection in the development of tuberculosis; this is seen especially in the ends of the long bones and in certain short bones. In osseous tuberculosis the epiphysis is the commonest seat of initial involvement, while in joints the synovial membranes become first infected. If the diseased bone is incised longitudinally there is seen, in the early stages of the affection, a small, yellowish, nodular mass within the cancellous structure at the epiphysis. Microscopic examination reveals numerous tubercles and bacilli. At a later stage of the disease the yellowish mass is found to have softened and to contain necrotic particles of bone. Still later, if treatment has been effectual, more or less fibrosis

of the parts and eburnation of the bone attest the fact that the condition is gradually healing. Subsequently much of the fluid is lost and the necrotic mass is of a dry, cheesy consistency. On the contrary, gradual destruction of the osteal and periosteal tissues may occur; and the necrotic material may be discharged through a sinus or fistula, which has developed. At times rupture takes place into the adjacent joint. In many cases, however, the joint is primarily affected upon its synovial surfaces. The process has the same general character as heretofore described; in addition, in the favorable cases extreme fibrosis gives rise to ankylosis or stiffening of the joint.

Symptoms.—The symptoms of bone and joint tuberculosis depend somewhat upon the point involved. The condition is typically chronic, beginning with vague pains and tenderness, together with gradual increase in the size of the part. If a joint is affected, stiffness slowly develops. Muscular spasm is often noted rather early in the disease; later there is considerable muscular atrophy. The latter condition serves to intensify the swollen appearance of the part. The cutaneous tissues are usually pale, and coursing over the part oftentimes prominent veins are seen. If treatment is effectual, no further changes may be noted, and the condition is likely to heal. If septic infection is added to the already existing tuberculous lesions, all the symptoms become intensified and the process often goes on to sinus formation, the pus from which contains many pyococci, and often but few, if any, tubercle bacilli. In spinal caries (Pott's disease), owing to the involvement of the bodies of the vertebræ, the process does not tend to extend toward the neighboring cutaneous surface, but rather down the sheath of the psoas muscle, and appears in the inguinal region. There is always more or less spinal deformity in Pott's disease (kyphosis, skoliosis). In hip-joint disease there is a tendency toward shortening of the affected limb.

Prognosis.—It is often well to give a guarded prognosis in cases of bone and joint tuberculosis in so far as the local condition is concerned. The possibilities of ankylosis must be kept in mind, for after considerable involvement of the structures has taken place complete restoration never occurs. Severe cases may give rise to widespread dissemination of the infection, and death is the sequel. Amyloid changes characteristically take place in the viscera (liver, spleen, kidneys, etc.) in consequence of the chronic tuberculous changes with suppuration.

General Diagnosis.—In addition to the aforementioned diagnostic procedures available in the several types and situations of tuberculosis, recourse may be had to tuberculin tests; agglutination tests also have been proposed, but they have not proved satisfactory. The tuberculin tests comprise: (1) The von Pirquet cutaneous reaction, which consists of preliminary scarification and the application to the scarified area of a drop of old tuberculin—in a manner analogous to that practised in ordinary vaccination; a scarified but otherwise untreated area is used

as a control. The test is of much value, especially in young subjects; in adults the importance of a slight reaction should not be overestimated, since many adults are the subject of more or less quiescent tuberculosis, of the bronchial lymph nodes, for instance; a marked reaction may be significant of reinfection or activation of a quiescent lesion. (2) The Moro cutaneous reaction, which consists of rubbing into the skin an ointment of old tuberculin and lanolin. The test is of somewhat less value than the von Pirquet. (3) The Wolff-Eisner or Calmette conjunctival test, which consists in instilling into the conjunctiva of one eye one drop of a 0.5 per cent. solution of old tuberculin. The ensuing reaction is sometimes so violent that the test deservedly has fallen into disrepute and disuse. (4) The hypodermic test, which consists in injecting 2 milligrams of old tuberculin; the reaction consists in the production, within twelve to twenty-four hours, of local irritation, malaise, neuromuscular pains or soreness, and fever (102° to 104°). Should the first injection prove unproductive, the injections may be increased to 3, 5, and 8 milligrams. Since the introduction of the von Pirquet test the hypodermic test has fallen into disuse. (5) Material (sputum, etc.) from a suspected case may be injected into a guinea-pig, and the pig killed at the end of four to six weeks (if it has not died earlier). In the event of the material having contained tubercle bacilli the animal will be found infected. (6) Ebbright has suggested injecting the suspected material into a guinea-pig; at the end of six days injecting 0.25 c.c. of old tuberculin; and killing the animal at the end of another twenty-four hours. In the event of tuberculosis, the bacilli may be found thus early, the view being that the large dose of tuberculin breaks down the body resistance and permits the rapid multiplication of the tubercle bacilli. (7) Yamamanchi has suggested injecting into a rabbit a few cubic centimeters of blood serum (or blister serum) from a case of suspected tuberculosis, and in a few days injecting tuberculin; the animal having become sensitized, experiences a severe, perhaps fatal, reaction. (8) The bacilli may be searched for in the circulating blood, as suggested by Rosenberger; but they are not likely to be found in many cases, assuredly not in early or localized infections.

Prophylaxis.—The control of tuberculosis may eventually be effected by prophylactic measures, of which the following are the most important: (1) The education of the public, by a continuation of the present-day widespread antituberculosis propaganda; by disseminating a knowledge of the efficient means of preventing the disease—into municipalities, industrial and other associations, schools, the family, etc. (2) Improved sanitary conditions under which, especially the poor, the industrial workers, etc., live and work. (3) Sanatoriums, hospitals, dispensaries, and social services for the study, prevention, and treatment of tuberculosis; especially important being the treatment of incipient and far-advanced and hopeless cases, and visiting the poor and ill-informed by trained nurses or social workers. (4) Supervision by the Public Health authorities, which should include notification of the disease (in a manner to avoid

wounding the sensibilities of the patients), the destruction of sputum, the disinfecting of rooms inhabited by tuberculous patients, supervision of dairies, abattoirs, etc. (5) Individual prophylaxis—which in general comprises the regulations laid down on page 29. In addition, children of tuberculous parents should have the benefit of the best environmental conditions, and assuredly should not sleep in the same rooms with their diseased parent or parents. The ideal conditions as regards future health suggest rearing the children apart from the tuberculous parent—virtually impossible of attainment, even were it in every respect desirable (which it is not). The child, however, should be out-of-doors as much as possible, have an abundance of good nutritious food, be well clad, and afforded a sufficiency of regulated exercises. In the event of illness, especially catarrhal disorders of the upper respiratory tract, enlarged tonsils, adenoids, enlarged cervical lymph nodes, general debility, asthenia and anemia, etc., efficient treatment should be instantly undertaken, and tonics, such as iron, quinine, strychnine, arsenic, cod-liver oil, etc., should be administered.

Treatment.—The efficient management of tuberculosis in general comprises the use of: (1) Fresh air, nutritious food, and regulated rest and exercise; (2) specific remedies; (3) medicinal agents, largely to combat symptomatic indications; and (4) certain additional measures applicable in special forms of tuberculosis, such as miliary tuberculosis, acute pneumonic tuberculosis, surgical tuberculosis, etc.

Fresh Air, Nutritious Food, Rest, and Exercise.—Nothing transcends in importance fresh air and nutritious food. The benefits of fresh air may be secured at home in the city or the country, in sanatoriums near at hand, or in certain mountainous or marine climates. If at home, the patient should be as much as possible out-of-doors, and he should sleep on a balcony or with the windows of his room wide open; he should, of course, be sufficiently, but not excessively clothed (as commonly obtains among the ignorant classes), and he should take the precaution so to arrange his bed that he is protected against continuous draughts, and the rigors of storms. Night air, dampness, rain, snow, and the continued low temperature of winter are not contra-indications to this open-air treatment; nor are cough, hemoptysis, fever, emaciation, etc. Excellent results have been achieved by the tuberculosis classes inaugurated by J. H. Pratt. It is not necessary, as a rule, for the patient to go long distances from home to secure the benefits of fresh air; indeed, this is positively contra-indicated in advanced cases; in those with strong home ties, in whom absence from home often causes marked mental depression; and in those to whom such distant travelling occasions financial hardships—which are not commensurate with the hoped-for advantages. Climate *per se* is not the major factor in the cure, that region being satisfactory that combines a pure atmosphere, considerable sunshine, and equable temperature (absence of sudden variations); other desirable features comprise dryness of the atmosphere (at least not excessive humidity), moderate altitude, and the facilities for good food,

good housing, and some amusement. Most, especially early, cases, with localized lesions, moderate fever, and little emaciation, and without excavations, do best at moderate altitudes (2000 to 2500 feet), such as the Adirondacks, Asheville, Aiken, etc. These afford the advantage that, as a rule, after the disease has been arrested the patient may return to the sea level without the likelihood of reactivation of the lesions. Some patients seem to do better at higher altitudes (5000 feet or more), such as Colorado, New Mexico, Arizona, Davos and other places in Switzerland, etc. Chronic and advanced cases, with excavation, fibrosis, emphysema, repeated hemoptysis, emaciation, etc., as a rule, do better at the sea level and in a moist climate, such as Florida, Bermuda, etc. Subjects with slowly progressing lesions are often much benefited by the dryer climate of Southern California, the Riviera, Egypt, Algiers, etc. The sanatoriums in these and other regions combine the advantages of strict disciplinary supervision of out-door life, rest, exercise, food, bathing, clothing, medicinal and other treatment; they are especially serviceable in early cases; and since they also exercise an educational influence not only upon the patients, but also among the people of their vicinity, their further establishment should be encouraged.

If the patient has fever, that is, if the highest daily temperature is above 99.5° to 100° F., he should be at rest in bed, or at least in a reclining (steamer) chair. In the absence of fever, properly regulated, supervised, and graduated exercise should form a part of the general management. This may consist of walking, graduated ascents (as is used in German sanatoriums), or various sorts of light out-door work, the results of which, that is, the reaction of the patient to which (temperature, pulse-rate, weight, etc.), must be carefully noted. These exercises, as a rule, are most advantageously prescribed in the morning hours.

Since the cure of tuberculosis depends upon the maintenance and improvement of nutrition, the greatest attention must be paid to the diet and the digestion. The food must be readily digestible, nutritious, varied, and appetizing, and it should be partaken of in stated amounts at stated intervals. An amount in excess of that which would be taken in health is necessary, but the excessive hyperalimentation in vogue several years ago seems undesirable. The diet should consist largely of milk, cream, butter, eggs, fish, bread, meats (especially beaf, mutton, chicken, and bacon), olive oil and other fats, etc. In addition to what might be considered a goodly amount of a varied diet, the patient should take daily from one and one-half to two quarts of milk and from four to six eggs. Many patients do best by taking for breakfast some fruit, a cereal, bacon, two eggs, and milk (hot, if desired, and flavored with coffee, tea, or cocoa); a full meal, for dinner, in the middle of the day, consisting, for instance, of soup, fish, meat, two or three vegetables, a salad, and fruit, a simple pudding or other dessert, or cheese; and for supper, in the evening, some cold meat, or fish, or two eggs, and toasted bread, boiled rice, or stewed fruit, and milk as at breakfast. The additional milk and eggs may be given between breakfast and dinner, between dinner and supper, and

just before retiring for the night; with the milk it is advisable to take several crackers or a small amount of toasted bread to assist in comminuting the curds. If the milk and eggs prove nauseating to the patient, they may be flavored, as already suggested, with coffee, tea, cocoa, or with chocolate, sodium chloride or bicarbonate, or a small amount of brandy, whiskey, port wine, sherry wine, etc.; and from time to time they may be discontinued for a day or two. Peptonization of the milk is sometimes serviceable for a time; or one may give kefir, koumiss, etc. Rarely it may be necessary to resort to Debove's method of forced feeding, which consists of preliminary washing of the stomach, followed by the introduction through the stomach tube of a quart of milk, one or two eggs, and three ounces of finely minced meat; this is done three times a day. A raw-meat dietary (zomotherapy), consisting of the use of one to two pounds or more of meat daily, either minced and raw, or made into a soup, has been readvocated lately. Anorexia and nausea are sometimes deterrents to proper feeding; but several days of life in the open air, or a change of scene, or an ocean voyage usually act as an efficient tonic to a jaded appetite. Sometimes, however, it may be necessary to administer tonics; but care must be exercised that medicines employed for other purposes do not derange the stomach. Alcohol, as a rule, is not indicated: indeed, most patients do better without it altogether; but there can be no serious objection to a small amount of brandy or wine to flavor the milk and eggs from time to time. In advanced cases with fever, emaciation, rapid pulse, night sweats, poor digestion, etc., alcohol seems to do good; at least it aids digestion, especially the digestion and assimilation of fats, stimulates the heart and the nervous system, promotes a feeling of well being, relieves the night sweats, induces sleep, and if it does not lead to fibrosis, as has been claimed, it does not accelerate the progress of the lesions.

Specific Remedies.—Tuberculin is a valuable adjuvant to other forms of treatment in selected cases. The preferable preparations are: Koch's new tuberculin, tuberculin residue (T. R.), consisting of the toxins and endotoxins of crushed tubercle bacilli unchanged by heat or chemicals; bacillen emulsion (B. E.), which consists of one part of finely pulverized virulent tubercle bacilli and one hundred parts each of distilled water and glycerin; and Deny's bouillon filtre (B. F.), which consists of a filtrate from a bouillon culture and contains all the soluble toxic and other products of the tubercle bacillus. The cases suitable for tuberculin treatment are the early cases with circumscribed lesions, in which there is little if any fever or emaciation, and no excavation, and chronic cases with good nutrition and no serious complications; distinct contra-indications comprise rapidly advancing lesions, hectic fever, rapid heart action, and progressive emaciation. Trudeau advises: to begin with small doses, such as $\frac{1}{10000}$ milligram (liquid measure) of the bouillon filtre (B. F.); to avoid provoking local or general reactions; to increase the dose gradually, at intervals of three or four days, or longer when large doses are given, until a maximum dose of 1 c.c. of the tuber-

culin is reached; in case a reaction occurs, especially if this be associated with fever and constitutional disturbances, to reduce the next dose to about half of that which induced the reaction; and to continue the treatment for months, but not indefinitely for fear of exhausting the patient's power to respond, by the formation of antibodies, to the stimulus of the injection: it being better to discontinue the treatment for from three to six months and resume it again if anything in the patient's symptoms or condition seems to make this necessary.

Medicinal Agents.—The medicinal treatment of tuberculosis is largely symptomatic; but certain remedies are given with the view to improve nutrition and increase the powers of resistance of the individual. Of these, the most valuable are arsenic, especially in the form of Fowler's solution, 5 minims (0.30 gram) three times daily; mercuric bichloride or succinimide, $\frac{1}{12}$ to $\frac{1}{36}$ grain (0.005 to 0.0018 gram) three times daily; strychnine, cod-liver oil, iron, the hypophosphites, etc. Inunctions of europhen (5 per cent. in olive oil) and other iodine preparations also have been commended.

While the symptoms of tuberculosis are commonly improved by fresh air, nutritious food, regulated rest and exercise, they sometimes demand special attention. Cough, unless excessive, need not be restrained medicinally; often it can be repressed mentally; since, however, it serves to remove secretion, it is to some extent a necessary evil. In general, if requiring treatment, it should be met with the measures mentioned under bronchitis. Creosote is a very valuable remedy, and may be given in doses beginning with 3 minims (0.18 gram) and gradually increasing, three times daily, in milk, hot water, brandy, or a bitter tincture. Guaiacol carbonate is sometimes better borne. The distressing nocturnal cough may be controlled by the following:

℞—Codeine	3 grains	0 20
Dilute hydrocyanic acid	24 minims	1 50
Glycerin	1 ounce	30 00
Cherry laurel water, sufficient to make	3 ounces	100 00—M.

S.—Two teaspoonfuls (10 c.c.) at 6 and 9 P.M., and during the night if necessary.

Comfort is sometimes promoted by diffusible stimulants, such as Hoffman's anodyne, spirit of chloroform, etc., or by hot whiskey on retiring; in other cases morphine may be required, but it should be given cautiously in the event of cavity formation, since the cavities must be emptied. Inhalations are often of marked benefit. Beverley Robinson extols the virtues of equal parts of creosote and alcohol, or, when there is much irritative cough, equal parts of creosote, alcohol, and spirit of chloroform (on an inhaler). Lees advocates the use of six to eight drops of the following on an inhaler, every hour during the day, and two or three times during the night, if the patient is awake:

℞—Carbolic acid	2 drams	8 0
Creosote	2 drams	8 0
Tincture of iodine	1 dram	4 0
Spirit of ether	1 dram	4 0
Spirit of chloroform	2 drams	8 0—M.

The treatment of hemoptysis is discussed on page 540. Pleural pains may be relieved by counterirritation with tincture of iodine to which a small amount of croton oil may be added; by a blister; by rubbing with a mixture of equal parts of menthol, camphor, and chloral; or by strapping the side. The night sweats may be controlled by an abundance of fresh air at night, and the avoiding of too many bed-clothes; by sponging the body with alcohol and alum water; and by the use of camphoric acid, 20 to 40 grains (1.3 to 2.5 grams), four hours before the time of the expected sweat; atropine, $\frac{1}{150}$ to $\frac{1}{100}$ grain (0.0004 to 0.0006 gram) at bedtime; picrotoxin, $\frac{1}{100}$ grain (0.0006 gram), aromatic sulphuric acid, agaracin, etc. In the event of fever, the patient should be at rest in bed. Rest in bed, fresh air, and nutritious food comprise the best treatment for the fever as such; but should the fever become high, attention should be paid to a sufficiency, but not an excess of bed-clothes; the body should be sponged repeatedly with cool water or with alcohol and alum water. Quinine, the salicylates, and the coal-tar products are distinctly contraindicated; of course, they may reduce the temperature, but at the expense of the bodily resistance. Painful dysphagia may be relieved by spraying the throat with cocaine (2 per cent.) or by swabbing the throat with orthoform (5 per cent.) in olive oil. The treatment of so-called dyspepsia and diarrhœa is similar to that mentioned elsewhere under these headings.

Special Forms of Tuberculosis.—In miliary tuberculosis, treatment is of very little avail. Supportive measures are indicated, especially strychnine, whiskey, camphor, etc., and in the event of restlessness, headache, and other cerebral symptoms, the bromides and opiates. In the event of meningitis lumbar puncture by relieving pressure may lead to amelioration of the symptoms; other measures mentioned under meningitis may be resorted to. The treatment of acute tuberculous pneumonia is that of pneumococcic infection of the lung, together with such of the aforementioned measures as may seem indicated when the tuberculous nature of the disease has been recognized. The treatment of many forms and localizations of tuberculosis is distinctly surgical: for instance, tuberculosis of the lymph nodes, of the serous membranes, of the skin, of the bones and joints, etc. Recourse has also been had to surgical intervention in certain cases of localized tuberculosis of the lung and with some encouraging results, details of all of which may be found in books of surgical practice.

DIPHTHERIA.

Diphtheria is an acute, specific, infectious, and highly contagious disease caused by Bacillus diphtheriæ (Klebs-Löffler), and characterized by fibrinous (croupous, or diphtheritic) inflammation of a mucous membrane, fever, and divers manifestations of toxemia.

Etiology.—Diphtheria is peculiarly a disease of childhood, occurring most frequently between the second and the fifth year, and attacking

the sexes equally (under similar circumstances). It is relatively infrequent after the tenth year, but adolescents and adults (nurses, physicians, attendants, etc.) are rather commonly affected and often succumb. The disease occurs in almost all parts of the world, but is especially prevalent in the temperate zone. It is endemic in most large cities, and frequently prevails epidemically, especially during the colder months of the year—the late fall, the winter, and the early spring; but the epidemics vary considerably in severity. In some sections the disease is relatively more common in the country districts than in the cities. General insanitary conditions are of etiological significance only in so far as they reduce the vitality of the individual and favor the harboring and multiplying of the bacilli. Overcrowding, such as obtains in schools, asylums, tenements, etc., is of significance in permitting the ready dissemination of the bacilli among large numbers of susceptible persons. One attack confers an incomplete and temporary immunity. Recurrences are not infrequent.

The exciting cause of the disease is Bacillus diphtheriæ, which may be isolated from the local fibrinous inflammations, the secretions and discharges from the throat and nose, sometimes from the blood and the internal organs, especially the lung (in the event of bronchitis and bronchopneumonia), but also the liver, spleen, kidney, etc. The disease is highly contagious and spreads with considerable rapidity. Infection may be transmitted by direct inoculation; by contact or proximity; by a third person (physicians, nurses, in whose hair the bacilli have been found, etc.); by fomites, especially room furnishings, room dust, bed-linen, personal linen, dishes, toys, etc., to which the bacilli often cling for a long time; by infected food and drink, especially milk (answerable for a number of epidemics), cheese, etc.; by domestic animals, etc. Infection is usually acquired by inhalation or swallowing, but it may also be acquired through a wound or abrasion. Individual susceptibility is of much etiological significance: many persons exposed to the infection do not acquire it, and others harbor the bacilli in their throats, and themselves escaping infection, often disseminate it. In the throats of convalescents, for instance, the bacilli may remain virulent for a long time (weeks and months)—so-called "chronic carriers," responsible for many outbreaks. Chronic nasopharyngeal catarrh, enlarged tonsils, carious teeth, etc., also favor infection.

Pathology.—The characteristic lesion is the so-called false membrane, the relative frequency of the situation of which is well exemplified by the results of the investigations of Councilman, Mallory, and Pearce, who, among 127 of 220 fatal cases, found it situated as follows: In the larynx, 75; trachea, 66; tonsils, 65; epiglottis, 60; pharynx, 51; nasal mucous membrane, 43; bronchi, 42; soft palate, including uvula, 13; œsophagus, 12; tongue, 9; stomach, 5; vagina, 2; duodenum, 1; vulva, 1; skin of the ear, 1; conjunctiva, 1. Thus, in fatal cases the larynx is most frequently involved; but this is not true of all, especially non-fatal cases, in the majority of which the tonsil is first and most frequently attacked. This false

membrane may be circumscribed in extent or widespread. Sometimes it is limited to a small region of the tonsil or soft palate; in other cases it covers the tonsils, soft palate, pharynx, larynx, and trachea; it may extend to the nose (which may become completely obstructed), to the conjunctiva (through the tear duct), and to the accessory sinuses (which, as pointed out by Councilman, Mallory, and Pearce, are very frequently involved, and the unhealed lesions of which are often answerable for the persistence of diphtheria bacilli in the nose and throat). It may travel through the Eustachian tube to the middle ear, over the tongue, and involve the oral mucous membrane, through the bronchi to their finest ramifications, through the œsophagus to the stomach, intestine, etc.; and by transference it may implicate other mucous membranes, such as the vulva, vagina, etc. In character, the membrane varies considerably: it may appear as a thin, whitish, more commonly grayish or fawn-colored, pellicle, easily removable without loss of substance or bleeding; or it may present all gradations of severity from this to a dense, tough, grayish, brownish, or blackish membrane, firmly attached in the early stages and separable only with difficulty and leaving a bleeding surface, but later softer and more shaggy; or the lesions may be markedly necrotic or really gangrenous. The membrane is more easily removed from the trachea and larynx than elsewhere, being sometimes expelled by coughing, and it rarely occasions in these regions a loss of substance extending beyond the superficial epithelium; the subepithelial layer, however, is always markedly injected.

Councilman, Mallory, and Pearce, who have studied the lesions attentively, describe them as follows: The earliest lesions are due to the toxin, produced by the diphtheria bacillus, possibly growing in the fluids of the mouth and throat. The toxin gives rise to degeneration and necrosis of the epithelium, often preceded by active proliferation of the nuclei of the cells by direct division. In this necrotic tissue, but more especially in the exudate speedily formed, the bacilli become implanted and there continue their deleterious action; but the bacilli themselves are not found growing in living tissue. "The cells may either break up into a detritus, with fragmentation of the nuclei, or they may become changed into refractive hyaline masses. An inflammatory exudation rich in fibrin factors comes from the tissue below, and fibrin is formed when this comes in contact with the necrotic epithelium. The fibrin in part is formed into a reticulum around exudation cells and degenerated epithelium, in part, it combines with the hyaline degenerated cells to form a hyaline membrane. It is probable that a hyaline membrane may be formed without the exudation; in this case the network of the membrane represents the edges of the cells, and the spaces the former nuclei. The hyaline membrane is most often formed on those surfaces which are covered with epithelium having several layers of cells. It may be formed by a hyaline degeneration of exudation cells; in this case the spaces in the meshwork are smaller. It is probable that the fibrinous membrane is formed both on the surfaces and in the tissue.

The fibrin is first formed around cells that afterward disappear. In the trachea the fibrinous membrane often has a definite structure. The membrane may disintegrate and be broken up into a mass of detritus (the process commences on the surface), or it may be cast off as a whole by being elevated by an exudation beneath. Very thick masses of membrane may be formed by the constant addition of fibrinous exudation. The membrane is never formed primarily on an intact epithelial surface, but it may extend over it. Nothing is to be gained by making an anatomical distinction between a croupous and a diphtheritic membrane. . . . The membrane formation is accompanied by changes in the tissue beneath, which represent a combination of degeneration and exudation. The connective tissue and bloodvessels undergo a hyaline fibrinoid degeneration very similar to the degeneration of the epithelium. Necrosis may extend deeply into the tissue, but there is little tendency to deep ulceration or abscess formation. The degeneration in the mucous glands of the tissue is so pronounced as to be almost specific. Marked degeneration of the epithelium of the glands may be found without any change in the surrounding tissue."

The organs in general show lesions common to most of the toxemias. In the kidneys the lesions vary from cloudy swelling to severe nephritis. In the liver the degenerative changes are not so marked, nor are they specific. The spleen is sometimes enlarged. The lymph nodes, especially those nearest the primary lesions, show changes—of two kinds: (1) Congestion, hemorrhage, and diffuse and circumscribed necrosis—found in many conditions; and (2) foci of proliferation, phagocytosis, and degeneration, resembling tubercles—distinctive of diphtheria, but found also in other conditions. Degeneration of the myocardium is common; it is usually fatty, sometimes hyaline. Acute interstitial myocarditis, endocarditis, and pericarditis also occur. Bronchopneumonia (acinous pneumonia, Councilman, Mallory, and Pearce) is found in more than half of the fatal cases. Peripheral neuritis (cranial and spinal) is common and answerable for many of the serious sequels. Poliomyelitis and hemorrhage into the cord and membranes are also encountered.

As already stated, general infection with the diphtheria bacillus (contrary to former opinions) is not uncommon, especially in the fatal cases; but in these cases there is usually also associated infection with the streptococcus (the most important and most common), the staphylococcus, the pneumococcus, etc. These organisms are also frequently found associated with the diphtheria bacillus in the local lesions in the throat and in the complications, especially the bronchopneumonias and the suppurations. These cases of mixed infection are often unusually severe clinically, and not infrequently terminate fatally—the one bacterium apparently assisting the other in producing the severe manifestations of the infection.

Variations in Virulence of the Diphtheria Bacillus—Avirulent Diphtheria Bacilli.—I have already said that the diphtheria bacilli may be found in the throats of healthy persons, and, sometimes for a long time, in the throats of convalescents. It is sometimes found also in throats in which the lesions are only a mild catarrhal tonsillitis or faucitis or a lacunar tonsillitis. The diphtheria bacilli, thus, vary considerably in virulence: some are slightly, if at all, virulent, that is, they produce little if any toxin (attenuated bacilli); others are more virulent, but the resistance of the individual is good and he escapes serious infection, though he may transmit serious and even fatal infection to others or give rise to a more or less widespread epidemic. In other cases bacilli are found that resemble the diphtheria bacillus more or less closely morphologically, but differ from it biologically—bacilli often ill-advisedly spoken of as the pseudodiphtheria bacillus, Bacillus xerosis (found in the healthy and diseased conjunctiva), and bacilli found also in many other places and conditions.

Diphtheroid Infections.—Whereas the diphtheria bacillus is the most common cause of pseudomembranous inflammation, it is absent in some cases, the causative agent being usually the streptococcus, occasionally the pneumococcus, the staphylococcus, and sometimes apparently other organisms ill understood and cultivated with difficulty, if at all, in artificial media (such as Vincent's bacillus, etc.). The preferable designation for these conditions is streptococcic, pneumococcic, or staphylococcic angina, tonsillitis, faucitis, etc. In view, however, of the occurrence of necrosis and fibrinous exudation they are not infrequently confounded with true diphtheria, and are sometimes spoken of as pseudodiphtheria, secondary diphtheria, diphtheroid, etc. Diphtheroid infection is the least objectionable of these, but it should be replaced by the other terms suggested. Thus, there is not infrequently a discrepancy between the clinical and the bacteriological diagnosis, some cases clinically diphtheria are provoked by other microorganisms, and certain other cases clinically very mild, with little, if any, membrane, are examples of true diphtheria. The many noteworthy additions to our knowledge during recent years, however, have led to a gradual revision of our conception of many morbid processes, and to the establishment of certainty and exact knowledge where formerly uncertainty and doubt were common; witness, for instance, the practical abandonment of the idea of "croup" as a distinct and separate entity.

These diphtheroid (streptococcic, staphylococcic, pneumococcic, etc.) infections or inflammations may occur: (1) As independent disorders, varying much in frequency, but sometimes, according to Park and Beebe, constituting as much as 40 per cent. of the then existing sore throats. They are only slightly (if at all) contagious; the symptoms, as a rule, are much milder than those of true diphtheria, and the mortality is much less—not more than 2.5 per cent. (Park and Beebe). (2) As secondary infections in several of the infectious diseases, especially scarlatina, measles, pertussis, typhoid fever, etc. The symptoms are

often severe, and through the supervention of general pyococcic infection not infrequently lead to death. Of course, true diphtheria also may complicate any of these or other infectious diseases.

Symptoms.—The period of incubation in true diphtheria varies from two to seven days, but in severe infections it may be scarcely more than twenty-four hours. As a rule, prodromes are slight or absent—consisting, when present, of general indisposition and disinclination to play, and possibly headache. The onset of the disease is usually gradual, the child complaining of increasing headache, apathy, general neuromuscular pains and aching, slight chilliness, and fever which soon reaches 102° to 103° F. Occasionally the onset is abrupt—with convulsions or vomiting and fever (103° to 104° F.). The subsequent local and general manifestations of the infection vary considerably, depending upon the virulence of the microörganisms, the localization of the process, and the local and general resistance of the patient.

In *pharyngeal or faucial diphtheria* there is usually more or less complaint of sore throat or of pain on swallowing; but in many cases the absence of such complaint is noteworthy. On inspecting the throat (a procedure that should never be neglected in sick children) the tonsils are found swollen, reddened, often peculiarly cyanotic, and a small patch of false membrane—whitish, grayish, or fawn-colored—may be visible. This forms, as a rule, first on one or the other tonsil, but soon involves adjacent structures. In some cases the pseudomembrane remains for some time undetected, having formed in regions not apparent to inspection, such as the posterior surface of the uvula, the recesses of the faucial pillars, etc. In these cases the disease, for the time being, is scarcely to be distinguished from an ordinary tonsillitis.

In mild cases the membrane remains confined to the tonsils, the uvula, and the soft palate. Frequently it may be removed with little loss of substance (or bleeding), but usually attempts to remove it result in exposing a raw-looking, bleeding surface—which soon becomes recovered. The fever is moderate (102° F.), the pulse somewhat accelerated (about 100 in children three or four years of age); the submaxillary glands are slightly, if at all, swollen, and the evidences of general intoxication are slight. There is usually a moderate leukocytosis and a trace of albumin in the urine. At the end of four to six days the fever begins to subside, and the membrane to shrivel, to curl up at the edge, and gradually to separate. By the eighth or ninth day it is usually gone, but the surface may remain red and inflamed for some time.

In cases of moderate severity, the false membrane is more widespread and deeper, inflammatory œdema and congestion are more marked, the submaxillary glands are considerably swollen, the appetite is lost, the fever is high (103° to 104° F.), the pulse is rapid (110 to 120), the urine is lessened in amount and contains albumin and casts, prostration is quite marked, and the child is more or less restless and delirious.

These cases not infrequently eventuate in death, but recovery may ensue at the end of ten days or two weeks.

In severe cases (malignant diphtheria) the child is usually quite ill from the beginning (but cases beginning mildly may become severe); vomiting, convulsions, and high fever may usher in the attack. The pharyngeal tissues become intensely œdematous, reddened, and swollen, the tonsils often meeting in the median line; the faucial tissues become covered with, apparently converted into, a grayish, brownish, or greenish-black, ragged, malodorous membrane or slough-like mass; a fetid, watery, or sanious discharge issues from the nostrils and the mouth; the submaxillary and other regional lymph nodes become much swollen and assist in quite fixing the jaw; the face, later the neck and chest, become swollen and œdematous, and in some cases a pallid lividity of the general integument supervenes. The fever may be quite high or remarkably low, even persistently subnormal (bad prognostically); the pulse is rapid, irregular, and weak, the respirations embarrassed and noisy (open mouth and obstructed throat); the urine is much diminished in amount and contains much albumin and many casts; and marked prostration, stupor, and delirium develop. An erythematous or purpuric rash is not uncommon; hemorrhages may issue from the nose or mouth, and contribute to a speedy fatal issue; or extensive sloughing and gangrene of the neck (Ludwig's angina) may ensue. Death usually results in from three to eight days from toxemia, cardiac and vasomotor paralysis, or certain complications.

Nasal diphtheria may occur as a primary disorder and later spread to the throat, or it may develop secondarily to faucial diphtheria. It may be mild, severe, or malignant in type. There may be little if any, membrane—only a profuse serosanguineous discharge, which quite naturally excoriates the upper lip (formerly believed to be of much diagnostic significance); or the membrane may be so marked as completely to occlude the nares. Glandular enlargement is usually marked (owing to the rich lymphatic supply of the nose), and the constitutional disturbances (prostration, albuminuria, delirium, etc.) are usually severe. In most cases of pharyngeal diphtheria the diphtheria bacillus may be cultivated from the nasal secretions, even in the absence of noteworthy lesions in the nose.

Fibrinous rhinitis is a curious disorder, subacute or chronic in course, characterized by recurring thick fibrinous exudation, benign in nature, and attended by slight constitutional disturbance—but due in 80 per cent. of the cases (Ravenel's collection of statistics) to the diphtheria bacillus.

Laryngeal diphtheria (formerly so-called membranous croup) develops usually secondarily to pharyngeal diphtheria, but it may occur as a primary disorder, and, as previously mentioned, laryngeal lesions are the most common in the fatal cases. In the majority of cases, the symptoms in the beginning are not unlike those of pharyngeal diphtheria —but the voice soon becomes hoarse, a rough, brassy, or "croupy"

cough develops, and the respirations become embarrassed. At the end of twenty-four or forty-eight hours, commonly at night at first, evidences of laryngeal or bronchial obstruction (stenosis) develop suddenly. At first these are often paroxysmal (spasm of the glottis), but later they become continuous (increasing mechanical obstruction). The inspiratory and expiratory efforts are attended by unusual efforts; they become loud and stridulous, and are associated with retraction (on inspiration) of the intercostal spaces and the epigastrium; the child becomes extremely restless and makes extraordinary efforts to get air, and becomes cyanotic and markedly prostrated. In the beginning these attacks sometimes subside spontaneously; in other cases they are relieved by coughing and the expelling of a portion of false membrane (sometimes casts of the larynx or a bronchus); in most cases, however, unless relieved, the dyspnœa and cyanosis become aggravated and continuous, restlessness, which at first becomes more marked, is gradually replaced by stupor and coma, prostration increases, and the child dies of asphyxiation. In some cases, in which the false membrane extends to the bronchi, death from suffocation may be comparatively slow. If the pharynx is not much involved, the evidences of toxemia at first are slight (on account of the poor lymphatic supply of the larynx), the fever is slight or moderate, and the general condition of the child is good —until the onset of the laryngeal obstruction. In laryngeal diphtheria, therefore, there is usually a marked disproportion between the severity of the local and the general symptoms.

Wound diphtheria occasionally occurs—when the diphtheria bacilli become implanted in a wound; but not all membranous inflammations of wounds are diphtheritic. Diphtheria occasionally occurs also on other mucous membranes, such as the conjunctiva, the vulva, vagina, anus, etc., and as a primary or a secondary infection.

Complications and Sequels.—The most important complications (due usually to pyogenic organisms associated or not with the diphtheria bacillus) are: Severe and even fatal hemorrhage from the nose and throat; bronchitis, bronchopneumonia (acinar pneumonia), and pulmonary atelectasis; suppuration of the lymph nodes; otitis media and disorder of the accessory sinuses of the nose; Ludwig's angina; cutaneous rashes (erythema, urticaria, purpura, gangrene); endocarditis; arthritis (simple and suppurative); suppression of urine and nephritis; general pyemia with metastatic abscess formation (secondary pyococcic infection). Diphtheria may also complicate certain of the infectious diseases, especially scarlatina, pertussis, measles, typhoid fever, tuberculosis, etc.

The most important sequels are the paralyses—which occur in about 20 per cent. of the non-fatal cases, are due to a toxic peripheral neuritis, and occur usually during the second or the third week of convalescence, though they may come on as early as the seventh or eighth day of the disease. The most common are: Paralysis of the palate (return of fluids through the nose, difficulty in swallowing, a nasal quality to the

voice, relaxation and impaired sensation of the palate); of the internal muscles of the eye (paralysis of accommodation); of the external muscles of the eye (ptosis, strabismus); of the facial muscles; of the laryngeal muscles (unilateral or bilateral); of the extremities or trunk (paresis, paresthesias, incoördination, loss of reflexes); and of the diaphragm. These usually subside in from two weeks to two months. The most serious paralysis involves the heart and the vasomotor system. This may result not only from toxic degeneration of the nerves supplying the heart, but also of the vasomotor nerves, and from toxic myocarditis, heart clot, etc. It may come on as late as two months after apparent convalescence, and may follow apparently mild attacks of diphtheria, though it is more common after severe attacks. Many of the deaths occurring during the course of diphtheria are due to this cardiac and vasomotor paralysis. The pulse is often slow rather than rapid; it becomes weak, the temperature sinks, the skin becomes pale, and the child soon dies. During convalescence death from this cause may occur suddenly, and entirely unannounced; it not infrequently follows immediately some unusual exertion. It is sometimes preceded by a warning arrhythmia, vertigo, vomiting, and signs of cardiac dilatation. Anemia often persists for a long time. Nephritis, laryngeal stenosis, chorea, etc., have been observed as sequels.

Diagnosis.—In many cases the diagnosis depends upon the demonstration of the diphtheria bacillus—which is conclusive. Provision for the ready and prompt determination of the bacteriological factor in cases of sore throat is now commonly made by the establishment of municipal and State laboratories; in the absence of these, private laboratories are usually available. But what is still better—the physician should train himself to make the necessary simple examination. A properly prepared cover-slip preparation will, in the majority of cases, within a few minutes convert doubt into certainty. Cultures also should be undertaken. Such examinations should be made in all cases of sore throat, since, as already mentioned, many cases mild clinically are truly diphtheritic and act as sources of infection. In the event of doubt all throat disorders in children should be regarded as diphtheritic, and the proper prophylactic and curative measures should be instituted immediately. Pending the detection of the diphtheria bacillus, the presence of an epidemic (or exposure to infection), the characteristic false membrane, a sanious discharge from the nose or the mouth, swelling of the submaxillary lymph nodes, and albuminuria are of much diagnostic value.

Streptococcic, staphylococcic, pneumococcic, and other pseudomembranous anginas (such as Vincent's angina, etc.) often can be excluded only by bacteriological examination; but in these the onset is usually more sudden than in diphtheria, the fever is higher and of shorter duration, glandular enlargement is less, albuminuria is often absent, there is less tendency for the lesions to spread to the nose and the larynx, and there is no history of exposure to diphtheritic contagion. In

scarlatina the throat lesions sometimes become "diphtheroid," and may not be distinguishable from those of true diphtheria except by bacteriological examination. Diagnostic difficulties are sometimes enhanced by an erythematous rash that occasionally occurs in diphtheria. However, this is uncommon, and is darker and of shorter duration than the true scarlatinous rash, and it is usually confined to the trunk. The two diseases not infrequently co-exist. In follicular tonsillitis the exudate is yellowish (rather than grayish or fawn-colored), patchy (corresponding to the follicles), is separated by intervening areas of congested red tonsillar tissue, does not tend to spread beyond the tonsils, may be removed without leaving a bleeding surface, and does not tend to reform (as in diphtheria). Spasmodic laryngitis, "false croup," "spasmodic croup" can scarcely be confounded with diphtheritic laryngitis ("true croup").

Prognosis.—The prognosis varies with the severity of the infection, the reaction of the individual, the presence or absence of complications, and especially with the treatment adopted. In some epidemics the death rate has been as high as 50 per cent. Since the introduction of antitoxin it has been reduced at least one-half; and in some large series of cases the prompt use of antitoxin has reduced the mortality to between 10 and 15 per cent. The prognosis is poorer in very young children, in the event of laryngeal and nasal involvement, marked toxemia, heart weakness, etc. The most marked discrepancies sometimes occur between the severity of the local lesions and the degree of systemic toxemia: minor local lesions are sometimes attended by very serious general manifestations; in some cases the patient is overwhelmed with the toxemia and dies within a day or two—before the local lesions have become noteworthy; while in still other cases well-marked and even extensive local lesions run their course without evidence of serious intoxication.

Treatment.—Isolation and prophylactic measures of the strictest sort should be carried out (page 29). Special attention should be directed to mild cases, ambulatory cases, convalescents (in whose throats virulent bacilli may remain for from six weeks to six months or more after the attack), and to attendants upon the sick. Catarrhal disorders of the nasopharynx, especially in children, should be corrected. Children exposed to infection should be required to use mild antiseptic nasal, oral, and pharyngeal washes (boric acid, potassium permanganate 0.5 to 1 per cent., mercuric bichloride 1 to 10,000), and should be given a prophylactic dose of antitoxin, 200 to 500 or 1000 units, depending upon its age.

The general hygienic and dietetic management of the developed disease is similar to that appropriate in the other infectious diseases in children. It is important, however, that the diet throughout should be fluid, concentrated, and readily digested. In the event of intubation or tracheotomy being necessary, rectal alimentation may be required.

The diagnosis of diphtheria brings with it the imperative indication for the use of the specific remedy—antitoxin. Even in case of doubt

as to the true nature of a throat disorder it is wise to administer diphtheria antitoxin—since in the event of the disease proving to be diphtheria the proper remedy has been administered at the most opportune moment, and in the event of its proving to be non-diphtheritic in nature, no harm has been done (indeed, possibly some good). The proper dose of antitoxin is that sufficient to insure the desired result; it is much better to give more than is really necessary than too little. In mild cases in children over two years of age, 3000 to 4000 units, and in severe (including laryngeal) cases 4000 to 8000 units should constitute the initial dose. This should be repeated in from six to eight hours if the swelling and congestion are not lessened, and the membrane seems to maintain its tenacious hold, and in from four to six hours, if the local lesions seem to be progressive and the membrane to be spreading. In some cases a third dose may be required at the end of four to eight hours, or after the lapse of twenty-four or thirty-six hours; and in a few cases, even a larger number of doses. Dr. McCollom, of Boston, has obtained the most gratifying results, in apparently hopeless cases, by using as much as 70,000 units. Large doses given early usually obviate the necessity of using antitoxin at the end of a week or thereabout after the first dose, and thus remove all possibility of producing anaphylaxia. The use of globulin preparations (from which some of the non-antitoxic serum substances have been removed) tends to lessen the likelihood of disagreeable rashes developing.

The antitoxin should be injected into the trunk or the thigh, and as early as possible (even pending the bacteriological report); but it is important to administer it no matter when nor how late the child comes under observation. The following statistics collected by Cohn illustrate the results of antitoxin treatment as influenced by the day on which it is commenced:

	Patients.	Per cent. mortality.
First day	78	1 3
Second day	361	11.1
Third day	284	10.5
Fourth day	101	24.7
Later and unknown	186	22.7

Striking results often follow the use of the antitoxin. When administered early, at the end of ten or twelve hours, the fever often abates, the local inflammatory œdema and congestion lessen, the pseudomembrane begins to curl up at the edge and soon loosens, and the evidences of toxemia diminish. In some cases, aside from anemia and weakness, the patient is quite convalescent in two or three days. The local lesions, however, are of more significance as suggesting the renewed use of antitoxin than the general manifestations. When the antitoxin is administered late, the results are not so striking, but the severity of the local lesions and the general infection are undoubtedly mitigated; many patients who otherwise would require it are spared intubation and tracheotomy; many in whom otherwise tracheotomy would be

required recover with intubation; the intubation tube may be removed much earlier than in pre-antitoxin days; and the mortality of intubation and tracheotomy cases is reduced one-half. The antitoxin, being a specific remedy, does not influence the lesions due to associated organisms (streptococci, staphylococci, pneumococci, etc.), nor does it prevent complications and sequels, except in so far as it influences and mitigates the severity of the primary diphtheritic infection.

Local treatment is advisable in most all cases, but it must be employed with discretion. The best treatment for the local lesions is the antitoxin internally; with no other remedy at our command are we so well able to destroy the diphtheria bacilli in the mouth, nose, and throat. Cleanliness, therefore, is the main object of local treatment. The best local treatment consists of irrigation through the nose or mouth, with a fountain syringe or a hand syringe, using physiological saline solution (than which nothing is better), or mild antiseptic solutions, such as boric acid; potassium permanganate, 1 per cent.; hydrogen dioxide, 10 to 20 per cent.; carbolic acid, 0.3 to 0.5 per cent.; mercuric bichloride, 1 to 10,000; formalin, 0.5 to 1 per cent., etc. These may be used every hour or two, depending upon the severity of the local lesions and the resistance of the child, remembering that the possible good that may be thus achieved may be more than counterbalanced by enfeebling results of the child's struggling. Sprays are likely to frighten the child, and are thus sometimes objectionable. Many physicians are partial to applications applied directly to the lesions on a swab; these often render good service, but should never be entrusted to other than trained hands—the physician or a trained nurse. The best preparation is Löffler's solution: Menthol, 10; toluol, 36; solution of iron chloride, 4; and absolute alcohol, 60. The more old-fashioned preparation, consisting of tincture of iron chloride, 3; carbolic acid, 0.5; glycerin, 15; and water, 15, is also excellent. Digestants such as papain and trypsin, etc., have been recommended, but I have not much faith in their efficacy. Gargles are of little use, since usually they do not reach the place where their action is desired.

In the event of laryngeal diphtheria, much good results from the inhalation (by means of a steam tent or croup tent) of steam, medicated with salt, lime, or compound tincture of benzoin, or the inhalation of the fumes of subliming calomel. External applications to the throat, hot and cold, are sometimes grateful, but they as frequently annoy and embarrass the patient as they help him. If symptoms of laryngeal obstruction develop, and the child is not too weak, the effects of vomiting at dislodging the false membrane may be tried: syrup of ipecac, ½ to 1 ounce (15 to 30 c.c.); tartar emetic, ½ grain (0.03 gram), hot mustard water, etc. If no relief follows, intubation should not be delayed, and it should always be undertaken if dyspnœa, cyanosis, and restlessness are increasing, and if retraction of the interspaces and of the epigastrium shows the difficulty the child experiences in breathing. If intubation does not afford relief, tracheotomy must be performed.

Alcohol seems to be a very serviceable remedy, especially when the infection is severe, the prostration marked, and the first sound of the heart has become weak. It may be given in doses varying from one to six ounces in the twenty-four hours. Strychnine also is valuable in some cases. The common heart tonics are not of much value, though apparently indicated on superficial observation; but in the event of collapse of the circulation they may be tried, especially the more diffusible stimulants. Previous to antitoxin days, tincture of iron chloride, calomel and other preparations of mercury, quinine, etc., enjoyed considerable reputation; they are sometimes valuable adjuncts, and are especially serviceable during convalescence (particularly the iron).

The complications are to be treated on general principles. Prolonged rest in bed (three to six weeks) during convalescence is essential.

INFLUENZA.

(*La Grippe; Grip; Epidemic Catarrhal Fever.*)

Influenza is an acute specific, infectious, and moderately contagious disease caused by Bacillus influenzæ (Pfeiffer), and characterized by fever, catarrh of the respiratory and the gastro-intestinal mucous membrane, severe neuromuscular pains, and marked prostration.

Etiology.—Although influenza is prone to attack adults, no age is exempt, and the sexes are affected about equally. While the most robust persons are frequently attacked, the disease finds its readiest victims among those debilitated by any cause, as well as among the aged, in whom it is frequently fatal. It usually prevails epidemically, especially during the late fall and the winter, and has been practically pandemic since 1889–90. The local epidemic visitation of the disease continues about six or eight weeks, during which time an extremely large proportion of the population may be affected. One attack does not confer immunity, since relapses are common. Some persons seem to be more or less immune naturally.

The exciting cause of the disease is Bacillus influenzæ, which may be found in the mucous discharges, and in the lungs and other organs in the event of complications. The disease is moderately contagious, infection being transmitted by direct contact, possibly by a third person, and by fomites; but the unusually large number of persons affected almost simultaneously is attributable to widespread aërial dissemination of the bacillus. In some epidemics the infection appears to have been spread by the ordinary channels of commercial intercourse. Infection is acquired by inhalation.

Pathology.—Influenza is devoid of characteristic lesions, the lesions found consisting of catarrhal inflammation of the respiratory and the gastro-intestinal tract, and rarely of enlargement of the lymph follicles of the intestine and of the spleen. The complicating lesions exhibit nothing specific.

Symptoms.—The period of incubation is usually two to four days. The onset is sudden, with severe chill or chilliness, high fever (often 104° to 105° F.), violent headache, especially about the eyes and the root of the nose, severe neuromuscular pains and tenderness, especially in the back and legs, catarrh of the respiratory mucous membrane, marked prostration, and depression of spirits. Depending upon the predominance of certain symptoms different types of the disease are described, but all gradations between the different types occur: (1) The respiratory type. In this, symptoms referable to catarrh of the respiratory tract are marked—coryza, conjunctivitis with excessive lacrymation, sore throat, hoarseness, harsh, dry cough. Sometimes the catarrh is more marked in, or confined to, the upper part of the respiratory tract, sometimes to the lower. Occasionally hemoptysis is observed. As a rule, after a couple of days there is a little mucous expectoration, and the usual evidences of bronchitis. As convalescence sets in the sputum becomes mucopurulent in character, but a harsh dry cough often persists for a long time. (2) Gastro-intestinal type. In addition to the general manifestations, nausea and vomiting sometimes occur as initial symptoms; later, abdominal pain and diarrhœa are common, and sometimes lead to collapse. Jaundice has been observed, and the spleen may be enlarged. (3) Nervous type. In this the catarrhal symptoms are relatively slight, but the neuromuscular pains, cutaneous hyperesthesia, sweating, and prostration are marked. Delirium and stupor sometimes supervene, and with continuation of the fever a typhoid state (typhoid type) develops. (4) A cardiac type, with feeble, rapid, and irregular pulse, sometimes progressing to serious cardiac embarrassment has been described. (5) A muscular or so-called rheumatoid type, with severe pains and tenderness in the back, extremities, and joints may occur. (6) A hyperpyretic type, and (7) an apyretic type also have been described.

In most cases the initial high fever remits at the end of twenty-four hours, and subsequently pursues an extremely irregular course, varying much in different cases. As a rule, it falls by lysis and reaches the normal in from three or four to eight days, but recurrence of the fever on slight provocation is quite common. With the decline of the fever, the severe neuromuscular pains, the headache, and the catarrhal symptoms abate and the patient gradually convalesces, though marked prostration and occasional sweatings may continue for a long time, and relapses are quite common. A second attack may occur during the same epidemic.

Complications.—Secondary infections are common. Although there is a true influenzal pneumonia, the frequent lobar pneumonia and bronchopneumonia are often due to mixed infection with the pneumococcus and the streptococcus. Abscess and gangrene of the lung and pleuritis have been observed. Myocardial weakness and irregularity of the heart action is common, and may last for a long time. Bradycardia also occurs. Endocarditis, pericarditis, and thrombophlebitis

are less common. Divers nervous complications, such as neuritis, meningitis, encephalitis, etc., as well as nephritis, otitis media, keratitis, herpes labialis and other cutaneous manifestations, etc., have been reported.

Treatment.—Rest in bed is the most important feature of the treatment, and should be insisted upon even in the mildest cases in the event of fever, since convalescence is much hastened by a few days' rest in bed, and the likelihood of complications and of sequels is much lessened. The patient should be isolated, if possible, and the mucous discharges should be disinfected. The diet should be light and easily assimilable. Aside from these measures the treatment is largely symptomatic, but should be always supportive. A warm bath or a hot mustard foot-bath, with an initial dose of Dover's powder (10 grains, 0.6 gram) and a hot drink at night, often serve to relieve the distress. Additional relief may be afforded by any of the following every two or three hours for the first day or two: aspirin, 5 to 10 grains (0.3 to 0.6 gram); quinine, 2 grains (0.13 gram), and salicin, 5 grains (0.6 gram); salol, 5 grains (0.3 gram), and acetphenetidin, 2 grains (0.13 gram), to which one may add small doses of Dover's powder, $\frac{1}{4}$ to $\frac{1}{2}$ grain (0.015 to 0.03 gram), or camphor monobromate, 1 grain (0.06 gram). Intense headache may be relieved by an ice-bag, caffeine, or the bromides. Rarely, especially in the event of very severe headache and of severe neuromuscular pains, it seems almost necessary to resort to the coal-tar products, but aside from the small doses of acetphenetidin already mentioned, their use must be attended by caution, since although they may relieve the subjective complaints, they only add to the already existing prostration. In many cases, stimulants are called for, of which the most valuable are whiskey, brandy, and especially strychnine, and the aromatic spirit of ammonium. The treatment of the catarrhal symptoms is similar to that suggested in acute coryza and bronchitis. The emunctories always require attention: mild diaphoretic remedies are valuable; the bowels should be opened freely; and the urine should be rendered copious by the generous use of water and mild alkaline diuretics. Especial care must be exercised in the cases of elderly subjects to prevent the development of complications, especially of bronchopneumonia, the cause of many fatalities in the aged. During convalescence strychnine is often required for a long time, and the persistent irritating cough without expectoration may require codeine or heroin.

BACILLARY DYSENTERY.

Bacillary dysentery is a specific infectious disease, caused by Bacillus dysenteriæ (Shiga), and characterized by abdominal pain, frequent intestinal discharges containing mucus and blood, and a specific serum reaction.

Etiology.—It is now generally believed that the common, especially acute, dysentery of the tropics and temperate zones is due to Bacillus dysenteriæ, an organism discovered by Shiga and later studied by Flexner and his pupils. Several strains of the bacillus are now recognized, that most common in this country being known as the Flexner-Harris. The especial habitat of the disease is the tropics, particularly Japan, the Philippines, Porto Rico, etc., where widespread and fatal epidemics are common; but sporadic cases occur elsewhere in the tropics and in practically the entire temperate zone, and epidemics are by no means infrequent, especially in the summer and fall. General insanitary conditions, such as may prevail in barracks, jails, asylums, etc., materially favor the spread of the infection, which also is one of the notable scourges of camp life and frequently numbers more victims than the military prowess of the enemy (our own Civil War, the South African War, etc.). How the infection is transmitted is not definitely known; the factors are believed to be similar to those of typhoid fever. The bacillus may be cultivated from the intestinal discharges and the blood, and from the intestinal contents and wall after death. In many cases they persist in the intestinal tract for a long time after apparent recovery, and may be cultivated from the intestinal discharges, either periodically or at all times: these cases constitute the so-called "chronic carriers," who are responsible for many epidemics. Amœba coli may be found associated with Bacillus dysenteriæ in some cases.

Dysentery is rather common also as a terminal event in certain wasting and debilitating disorders, and similar symptoms are observed sometimes to follow the ingestion of unwholesome food, especially unripe or overripe fruit, certain preformed poisons, etc. The summer diarrhœas of children also are often due to Bacillus dysenteriæ infection.

Pathology.—Two types of the disease may be recognized, the acute and the chronic. In acute cases ending fatally within a week, the mucous membrane of the large intestine is markedly swollen, œdematous, and hyperemic, and covered with blood-stained mucus, the solitary follicles are enlarged, foci of hemorrhage are present in the mucosa and submucosa, and the superficial layer of the mucosa (in folds, irregular patches, or larger areas) is necrotic and may be readily removed. In more severe infections the entire wall of the large intestine is very much thickened and stiffened (œdema, congestion, and cellular infiltration), and the mucosa is grayish black in color—necrotic or gangrenous. Even the serous coat may be involved, and sometimes the ileum shows more or less well-advanced lesions. The changes in the mucosa consist of coagulation necrosis with the exudation of fibrin and polynuclear leukocytes (pseudomembrane). The submucosa shows hemorrhages, fibrin formation, and irregular cellular accumulations (chiefly plasma cells); these are believed to be toxic in nature, since they may be present with an intact mucosa, and bacteria, as a rule, are not encountered amidst them. Ulceration does not occur, any loss of tissue resulting being due to exfoliation of necrotic mucosa and attached pseudomembrane. Should

the patient not die, the acute lesions may subside or they may become chronic. Of the chronic infection, follicular, diphtheritic, and gangrenous forms are sometimes described—which, however, are merely stages of the one disorder. In the milder follicular type there is necrosis and ulceration of the solitary follicles, with the gradual extension of the processes to the mucosa and submucosa and sometimes the muscular coat—whence superficial and often deep ulceration results (doubtless, in part at least, attributable to secondary, especially pyococcic, infection). A croupous or so-called diphtheritic membrane is present in many cases; it is usually irregularly distributed, and associated with more or less extensive sloughing, so that a characteristic shaggy appearance is produced. In other cases, more or less widespread gangrene occurs. Healing may ensue, but it is usually extremely slow, being much retarded by the secondary pyococcic invaders; as it occurs, irregular cicatrices develop which not only replace the mucous membrane, but, contracting, give rise to considerable distortion and even obstruction of the intestines. Polypoid outgrowths also are a feature of many chronic cases. The specific microörganism may be isolated from the lesions, especially during acute exacerbations—which are quite prone to occur.

Symptoms.—The period of incubation is believed to be, as a rule, less than forty-eight hours, and the onset of the disease rather sudden—with abdominal distress, frequent bowel movements, and fever (101° to 102° F.). In other cases the onset seems less sudden, the dysenteric symptoms developing out of what appears to be an ordinary diarrhœa. In whatever way developing, the bowel is soon rid of fecal matter, and although small amounts may still be passed from time to time, the stools consist largely, if not exclusively, of mucus admixed with blood and sometimes with pus, and in aggravated cases shreds of necrotic mucous membrane or even larger sloughs of the bowel. The stools are small in amount and very malodorous; they vary in number from ten to thirty, forty, or more in the twenty-four hours; they are usually accompanied and followed by extremely painful and distressing tormina and tenesmus. The abdomen is distended and often tender along the course of the colon. The constant straining may result at first in spasm and later paralysis of the sphincter, and even prolapse of the rectum. Evidences of general infection are seen in the fever, which may increase to 103° to 104° F. in the first few days, headache, occasional vomiting, loss of appetite, coated tongue, thirst, scanty albuminous urine, and rather rapid emaciation. In very severe infections, the rectal tenesmus and efforts at stool may be almost constant, the bloody mucous stools may be replaced by more serous discharges, abdominal distress and colonic tenderness may become extreme, delirium and the typhoid state (toxemia) may supervene, and in collapse the patient may die within three or four days. In the majority of cases, however, the stools lessen in number in from four to six days, the tenesmus subsides, the fever disappears, and the patient begins to improve generally, though some blood and mucus may persist for several weeks. In some cases improvement is arrested after ten days

or two weeks, and the general nutrition of the patient begins again to fail; he becomes anemic and emaciated, his pulse becomes weak and small, his temperature perhaps subnormal, and he passes into chronic dysentery. He may have three or four stools in the twenty-four hours—partly fecal, partly mucous, and sometimes admixed with blood, and acute exacerbations may occur from time to time; or constipation may alternate with diarrhœa. Recovery may finally ensue, or death may eventuate from exhaustion or some intercurrent infection.

Complications and Sequels.—Peritonitis sometimes occurs, but it is due rather to extension through the wall of the intestine than to perforation; perityphilitis or periproctitis is the most common. Inflammation of the other serous membranes, arthritis, pylephlebitis, etc., have been observed. Anemic œdema, amyloid disease, neuritis (with paraplegia and other paralyses), chronic nephritis, pyemia, intestinal obstruction, etc., may follow chronic dysentery.

Diagnosis.—The diagnosis is usually quite apparent from attention to the stools. The nature of the infection may be ascertained by recourse to the serum agglutination test with the dysentery bacillus (the Shiga organism, but especially the Flexner-Harris type in this country); by cultivating the organism from the stools; and by the deviation- or fixation-of-the-complement test.

Prognosis.—The prognosis depends, as a rule, upon the severity of the infection. The acute forms, especially when occurring in epidemics, are very fatal. In milder cases the persistence of blood and mucus is unfavorable. As a secondary infection in debilitated subjects, dysentery often determines the fatal outcome.

Treatment.—The prophylactic measures advised in connection with the infectious diseases (page 29), especially typhoid fever, should be carried out, since there is reason to believe that the factors in infection are similar to those of typhoid fever. In particular, the stools should be rigorously disinfected.

Rest in bed during the acute stage is essential. The diet must be the blandest—milk (peptonized, or diluted with barley water, lime water, or Vichy water), albumen water, raw eggs, beef juice, broths, custard, milk toast, etc. In subsiding or chronic infections the greatest attention also must be paid to the diet, since recurrences are not infrequently provoked by dietetic indiscretions. It is wise at the beginning of the attack, if there has been little diarrhœa, to clear out the bowel with blue mass, calomel, or castor-oil to which 20 minims (1.25 c.c.) of tincture of opium may be added. Following this, one should prescribe so-called intestinal antiseptics and astringents, such as:

℞—Bismuth subnitrate (or subgallate)	3 drams	12	0
Salol	1 dram	4	0
Deodorized tincture of opium	2 drams	8	0
Chalk mixture, sufficient to make	3 ounces	100	0.—M.

S.—Two teaspoonfuls (10 c.c.) every two or three hours, as required.

Bismuth salicylate, beta-naphthol, resorcin, creasote, zinc sulphocarbolate, and similar drugs are also useful; or one may give mercuric bichloride, $\frac{1}{100}$ grain (0.0006 gram), every two hours. In other cases, the following often leads to marked amelioration of the symptoms :

℞—Lead acetate	20 grains	1|3
Powdered opium	5 grains	0|3
Camphor	10 grains	0|65

Mix and make ten capsules.
S.—One capsule every two or three hours, as required.

In some severe cases of undetermined or supposed amœbic nature, recourse may be had to the ipecac treatment mentioned under amœbiasis (page 177). Irrigation of the bowel is of very great advantage in many cases, but the pain and tenesmus are often almost insurmountable obstacles to its use in acute cases; but sometimes these may be at least partially overcome by a preliminary enema of laudanum ($\frac{1}{2}$ to 1 dram, 2 to 4 c.c.) and thin starch water (2 to 4 ounces, 60 to 120 c.c.), or a cocaine or iodoform suppository. The irrigation should be done by means of a fountain syringe and the tube should be inserted high up in the sigmoid; a double catheter may be employed, so as to facilitate the exit of the fluid; and the fluid should be quite cold or quite hot (110° F.). From two to four pints (1 to 2 liters) of any of the following solutions may be employed: Silver nitrate, 0.1 to 0.5 per cent.; zinc sulphocarbolate, 0.1 to 0.3 per cent.; lead acetate, 0.1 to 0.3 per cent.; and salicylic acid, 0.2 to 0.5 per cent. In chronic cases, in addition to attention to the diet, large doses of bismuth, intestinal antiseptics, a lead and opium pill, and irrigations, especially with silver nitrate (1 to 5 per cent., followed by injection of sodium chloride solutions), should be tried. In some chronic cases rebellious to medical treatment, appendicostomy with subsequent irrigation of the colon has been followed by much benefit. A satisfactory antidysenteric serum has not yet been produced.

TETANUS.

(*Lockjaw.*)

Tetanus is an acute, specific, infectious disease caused by Bacillus tetani (Nicolaier), and characterized by increased reflexes and tonic spasms of the voluntary muscles.

Etiology.—The tetanus bacillus is widely distributed in nature, being found in the intestine of herbivorous animals, in manure, in the soil, in dust, in rust, in gun-powder, etc. An essential factor in the etiology of the disease is a wound—whence infection occurs; since, although two varieties of tetanus are described, the so-called idiopathic and the traumatic, careful observation is reducing materially the supposed idiopathic cases. These are said to develop after exposure to the inclemencies of the weather, especially after sleeping on damp ground, but small abrasions cannot be excluded in these cases. In other

cases, doubtless, insignificant wounds of the nose or the mouth afford ready entrance for the tetanus bacillus. The obviously traumatic cases follow wounds, especially contused and punctured wounds of the hands and feet, but also gun-shot wounds of any part of the body; they are not (or were not) infrequent in the newborn (tetanus or trismus neonatorum), infection occasioned by filthy dressings occurring by way of the umbilicus; and they sometimes occur in puerperal women, infection occurring by way of the genital tract. The disease is said to be more common in the negro than in the white race, and in the tropics than in the temperate zone—doubtless, however, merely in consequence of accidental factors.

The tetanus bacillus, having gained access to a wound, develops and, especially when associated with pyogenic cocci, produces a toxin of extreme activity (a fractional part of a milligram having been estimated to be a fatal dose). This is taken up by the lymphatics of the nerves and conveyed to the central nervous system, where it enters into such firm combination with the nerve cells that it frequently cannot be dissociated nor its effects overcome—although a potent antitoxin can be produced in the lower animals. The tetanus bacillus may be isolated from the wound, and recently has been found also in the lymph nodes adjacent to the infected wound.

Pathology.—Irregular congestions of the central nervous system, degeneration of nerve cells, and swelling and œdema of the peripheral nerves are encountered, but the lesions are in no way characteristic.

Symptoms.—The period of incubation is from five to fifteen days, as a rule. The shorter the period the more severe the symptoms and the more likely death. Generally there are no noteworthy prodromes. Preceded sometimes by general malaise, headache, occasionally chilliness, the disease begins with gradually increasing stiffness and tension of the muscles of the jaw and the neck, and difficulty in mastication. In a few cases the first manifestations are in the muscles near the site of infection. Gradually the tonic spasm involves almost all the voluntary muscles of the body: the jaws become locked, immobile; the forehead becomes wrinkled, the eyelids partly closed and the eyes staring, and the angles of the mouth drawn down and out (risus sardonicus); the head becomes retracted; spasm of the spinal muscles causes arching of the vertebræ (opisthotonos). The abdominal muscles become rigidly contracted; sometimes such is the contraction that the body may be arched forward (emprosthotonos) or rupture of the muscle may occur. The body is rarely arched to the side (pleurosthotonos). The legs and the trunk are sometimes extremely rigid (orthotonos), the patient being, as it were, held in a vice. The arms, as a rule, are relatively unaffected. Occasionally strabismus develops. The persistent tonicity of the muscles is made worse by paroxysmal exacerbations, which, induced by the slightest jar or noise, recur at irregular intervals, last from ten to thirty seconds, are accompanied by profuse perspiration, and occasion the greatest agony. The distress is frequently augmented by spasmodic dysphagia,

and by spasm of the muscles of respiration, of the diaphragm, and of the glottis, causing asphyxia which may prove fatal.

The mind is usually clear, the superficial and the deep reflexes are greatly exaggerated, and the patient is often wakeful (but the spasms often lessen during sleep). The pulse is rapid (100 to 140). The temperature may be normal, but it is usually somewhat elevated; hyperpyrexia is common in fatal cases (108°, 110°, 112° F.), and may even increase after death. The urine may be suppressed, or difficulty in voiding urine secreted may be experienced. Occasionally slight albuminuria or glycosuria is found.

Acute and chronic, severe and slight, cases may be distinguished. In the acute cases the phenomena develop rapidly and soon become intense, paroxysm following paroxysm with rapidity; death usually follows within three or four days from exhaustion, asphyxia, or heart failure. In more favorable cases the paroxysms become less frequent and less severe, and the patient may recover after the lapse of several weeks, though exhaustion may last for a long time and eventually prove fatal.

A special form is the cephalic tetanus of Rose, which follows injuries to the cranial nerves (especially the face), and is characterized by paralysis of the muscles supplied by the facial nerve on the side of the injury, and spasm of the masseters and of the muscles of deglutition (tetanus hydrophobicus), and sometimes of the muscles of the neck and the trunk.

Diagnosis.—The disease is usually unmistakable. In strychnine poisoning, as contrasted with tetanus, there is often a history of the ingestion of a supposed poison; the spasms are violent from the beginning, but do not begin with trismus, and are marked in the extremities (relative freedom of the arms in tetanus); there are intervals of complete relaxation (as contrasted with the persistent tonicity of tetanus); and strychnine may be found in the gastric contents. Tetany is often associated with rickets or gastric dilatation; the spasms involve the extremities, especially the hands and the feet and the larynx; and Erb's, Chvostek's, and Trousseau's signs may be elicited. In hydrophobia there is a history of the bite of a supposedly rabid animal; the spasms involve especially the muscles of deglutition and of respiration; there are marked psychic symptoms; and there is no lockjaw, no persistent tetanic spasm, and no opisthotonos. Hysteria occurs especially in neurotic women; the spasms may begin suddenly and last for hours or days; laughing and crying spells are common, especially upon the relaxation of the spasm; and other phenomena of hysteria may be elicited.

Prognosis.—The mortality of acute traumatic tetanus, of puerperal tetanus, and of tetanus in the newborn is over 90 per cent. In the subacute or chronic cases the mortality is not more than 50 per cent. Favorable symptoms are late onset of the symptoms (two weeks or more after the injury), more or less restriction of the spasms to the jaw and neck, lessening of the paroxysmal exacerbations, and the absence of fever.

Treatment.—The patient should be kept absolutely quiet in a darkened and secluded room, free from all irritating noises and solicitous friends. The diet must be light and nutritious, and administered in small amounts at frequent intervals. Should the patient be unable to swallow, a stomach tube may be passed through the nose or mouth (space left by absent teeth) and the patient thus fed, or recourse may be had to rectal alimentation. Inasmuch as the toxin is manufactured in the local wound, wisdom suggests its removal; should this be impossible the wound must be thoroughly cleansed and antisepticized.

The tetanus antitoxin has not sustained the early claims made for it, not so much, however, because it is impotent as because by the time it is generally used the tetanus toxin has already effected such a firm combination with the nerve cells as to prohibit all efforts at its dissociation. It is imperative, therefore, that the antitoxin be administered at the earliest possible moment, and in large amounts (2000 to 7000 units). It should be injected into and subcutaneously about the wound, and it may also be injected into the nerve trunks leading from the infected area, where perhaps it may meet and neutralize the toxin, being thus conveyed to the central nervous system. The antitoxin has been administered to a fairly large number of persons (mostly children) injured with toy pistols (Fourth-of-July celebrations) with a marked reduction of the morbidity incidence and the mortality. In view of the uncertain results attending the subcutaneous and intravenous injection of the antitoxin, its injection beneath the cerebral membranes or directly into the brain tissue has been practised—with improved results. This seems to me a rather unnecessary and hazardous proceeding, especially in view of the fact that the same object can be achieved by injecting it into the subarachnoid space by means of lumbar puncture. One should first assure himself that he has penetrated the arachnoid membrane, by withdrawing a considerable amount of the cerebrospinal fluid, and should then inject the antitoxin (30 c.c.) and subsequently more or less invert the body so as to insure the thorough mixing of the antitoxin with the remaining cerebrospinal fluid. The procedure should be repeated in accordance with the exigencies of the case. Meltzer, Miller, and others recently have obtained good results from the subarachnoid injection of a 25 per cent. solution of magnesium sulphate, of which from 1 to 2.5 c.c. may be used daily or every second day, depending upon the indications. Baccelli speaks highly of the hypodermic use of carbolic acid—in 2 per cent. solution, from 3 to 5 grains daily. I have thought that I have seen it do good. The spasms are best controlled by the use of chloroform and morphine, either or both of which should be given in sufficient dose. Chloral and the bromides are useful adjuvants. Stimulants (alcohol, digitalis, caffeine, etc.) are often called for to combat exhaustion and to maintain the failing heart. Cannabis indica, calabar bean, belladonna, curare, amyl nitrate, etc., have also been recommended.

CHOLERA.

(*Asiatic Cholera.*)

Cholera is an acute, specific, infectious, and moderately contagious disease caused by Microspira comma (Koch), and characterized by vomiting, profuse "rice-water" intestinal discharges, suppression of urine, collapse, and frequently death.

Etiology.—Cholera is endemic in India and has occasionally prevailed epidemically in Europe and this country. It always follows the channels of commercial intercourse (immigrants, pilgrims, shipping, etc.), and is in consequence more common in sea-ports than in inland towns. Age, sex, and race are of no etiological importance. Persons debilitated from any cause, the impoverished, alcoholics, subjects of chronic gastro-intestinal catarrh, mental depression, etc., seem especially susceptible. Some persons (50 per cent. of the population, Koch) are apparently naturally immune. One attack confers a temporary immunity.

Infection is spread especially by water—that used for drinking purposes, that used in preparing foods, such as milk, lettuce, and other foods eaten uncooked, and that used in domestic washings, etc. Infection, thus, is acquired by ingestion. Flies seem to be active agents in disseminating the infection. The disease is only slightly contagious. Infection is not transmitted by the air (the comma bacilli die soon when dried), but some persons have become infected from handling infected bed-linen, dishes, etc., and several cases of laboratory infection have been observed.

Pathology.—In consequence of the great loss of fluids through vomiting and diarrhœa, the body after death is shrunken and withered. Post-mortem rigidity comes on soon, is extreme (occasioning distortion of the body), and persists for a long time. Postmortem twitchings of the muscles (causing movements of the dead body) and even elevation of the temperature have been observed. The blood is thick and dark-colored, and the organs, as a rule, are unusually free of blood and dry. The important lesions are in the intestinal tract. The peritoneum is dry, though sometimes curiously sticky, and the coils of intestine are shrunken and thin. The intestinal contents resemble the "rice-water" discharges. The mucous membrane at first is swollen and congested and covered with an excessive secretion of mucus; later the lymphoid follicles become swollen, and may reveal irregular foci of congestion, occasionally of hemorrhage; the epithelium desquamates and may even be shed in rather large masses, leaving the submucosa denuded. Occasionally extensive necrosis and ulceration, rarely perforation, may be observed. The mesenteric lymph nodes are often swollen; the spleen is not enlarged. The other organs manifest the lesions common to most toxemias, modified, however, by the unusual loss of fluids. The comma bacilli may be found in the contents of the intestine

and in the mucous membrane; in protracted cases they may be found in the wall of the intestine and even in the mesenteric lymph nodes.

Symptoms.—The period of incubation is from one to three days. In some cases a premonitory stage may be distinguished—in which there may be some epigastric distress, diarrhœa, and weakness. Often, however, the onset of the disease is abrupt, coming on apparently in the midst of perfect health, with chilliness, profuse diarrhœa, and extreme prostration or collapse (stage of collapse). The stools are at first fecal in character, but they soon become grayish, alkaline, watery, odorless, and contain flakes of mucus, epithelial cells, and granular debris ("rice-water" stools), occasionally blood, and they usually reveal the comma bacillus in pure culture. There may be thirty or more stools within the first twenty-four hours. Usually they are passed painlessly, but occasionally there is complaint of abdominal pain and tenesmus. Vomiting soon comes on and becomes persistent, thirst is extreme and very distressing, painful cramps develop in the muscles of the abdomen and legs, prostration and collapse become more marked, and the toxemia and concentration of the blood increasing, the patient passes into the algid stage. The cutaneous temperature is much below normal and the skin feels cold and clammy, though the internal (rectal) temperature may be 103° to 105° F.; the extremities are cyanotic, the face grayish, ashen, or exhibits a slight cyanotic tint; the features are pinched, the eyes and cheeks sunken, and the skin in general withered and wrinkled. The pulse becomes weak, the heart sounds indistinct, the blood pressure low, the voice hoarse and weak, the breathing labored and superficial (asphyxic stage), and the urine much reduced in amount, and contains albumin, casts, and erythrocytes; not infrequently it is totally suppressed. Consciousness is often maintained throughout, but the patient may pass into coma, with or without delirium, and die—sometimes within the first twenty-four or forty-eight hours.

In some cases, however, the patient improves and the stage of reaction (tepid stage) sets in. The vomiting ceases, the intestinal discharges lessen and become more fecal in character, the pulse becomes stronger, the respirations easier, slower, and deeper, and the surface temperature normal; not infrequently the skin is covered with sweat, and the functions of the kidneys are gradually restored, though albuminuria persists for some time. In favorable cases the patient continues to improve, and convalescence is established at the end of ten days or two weeks. Not infrequently, however, diarrhœa recurs at the end of two or three days, the patient passes rapidly again into the algid stage, and soon dies. In other cases so-called *typhoid cholera* develops; the patient becomes delirious and often feverish, and the pulse rapid and weak; the urine is again suppressed, coma and perhaps convulsions supervene, and death usually ensues, though recovery has been observed.

Varieties.—Some attacks are so mild that the patient is slightly, if at all, indisposed, complaining only of a little diarrhœa; indeed, during epidemics the cholera bacillus has been found in the stools

of presumably healthy persons. The term *cholerine* has been applied to cases, resembling, if not identical with, *cholera nostras* (due to bacteria other than Microspira comma), in which there is profuse diarrhœa, possibly vomiting, but only moderate collapse, and rather quick recovery. In other cases the patient is overwhelmed with the intoxication and dies within a few hours, in coma, even before the onset of diarrhœa (*cholera sicca*). As a rule, the symptoms are directly proportional to the loss of fluid and the salts of the blood.

Complications and Sequels.—The most common are: protracted convalescence and anemia, nephritis, uremia, cutaneous rashes (roseola, erythema, urticaria), pneumonia, arthritis, parotitis, chronic enteritis and colitis, gangrene, especially of the toes, etc.

Diagnosis.—In the presence of an epidemic it may be difficult or impossible to distinguish between cholera asiatica and cholera nostras, except by a bacteriological examination of the intestinal discharges. The "rice-water" discharges of the early days of true cholera usually reveal the cholera bacillus in pure culture; later certain contaminating bacteria are usually encountered; while still later the cholera bacillus may be detected only with difficulty. The agglutination test may prove of service. Certain forms of food-poisoning, of fungus-poisoning, and of metallic poisonings (arsenic, antimony, and corrosive sublimate) may simulate cholera, but the associated phenomena usually preclude diagnostic error.

Prognosis.—The mortality varies from 50 to 80 per cent. in different epidemics, and is in direct ratio to the severity of the infection. Most deaths occur during the algid stage.

Treatment.—Prophylactic measures are of the greatest importance, and the strictest sanitary measures must be enforced (page 29). As in the past, rigid quarantine will prevent the introduction of the disease into this country. Haffkine's preventive inoculations have proved of some service, and seem to merit continued trial. During the prevalence of an epidemic all drinking water and milk should be boiled, and no uncooked food should be eaten. Dilute sulphuric acid, 20 minims (1.25 c.c.), in a pint (500 c.c.) of water, three times daily, is believed to be a serviceable prophylactic, since dilute acids kill the vibrios. All diarrhœic attacks of whatsoever nature must be promptly treated—by preliminary cleansing of the bowel (castor oil), and by astringents, such as bismuth, lead acetate, aromatic sulphuric acid, and opium.

When the disease has developed the patient must be kept at rest in bed in a warm room (to help maintain the bodily heat), and upon the development of the algid stage, he should be surrounded by hot bottles, etc. Morphine or Dover's powder is useful in the early stage in allaying the pain; hot applications to the abdomen (mustard plaster, spice poultice, turpentine stupe, etc.) are grateful. In the event of vomiting, small doses of calomel should be administered; most other remedies, such as cocaine, are useless. Efforts may be made to restrain the diarrhœa (aromatic sulphuric acid and astringents), but unfortunately our

best directed efforts are usually in vain. Enteroclysis with hot physiological saline solution, to which tannic acid (1 per cent.) and tincture of opium have been added, may be tried. Intestinal antiseptics seem to be indicated on theoretical grounds, and good results have been claimed for salol; naphthalene, resorcin, etc., may also be tried. The most efficacious treatment appears to be that recommended by Rogers and others—the intravenous injection of hypertonic (1.35 per cent.) sodium chloride solution: four drams (16 grams) to a quart (liter) of water. Of this two liters should be injected at a time, and repeated as occasion may warrant. Rogers adds to the first and second injection fluid, calcium chloride, 6 grains (0.4 gram) to the quart (liter), to overcome the low coagulability of the blood. The fluid may also be injected into the peritoneal cavity; and this may be augmented by hypodermoclysis and hot enteroclysis (107° to 110° F.). If the patient is not vomiting, fluid should also be given by the mouth. Strychnine, whiskey, brandy, camphor, hot coffee or tea, and more diffusible and rapidly acting stimulants, such as aromatic spirit of ammonium, and compound spirit of ether, etc., are also indicated in most cases. The uremic manifestations, which are likely to develop during the reaction stage, are associated with low blood pressure (80 to 90 mm. Hg.), and are to be combated by the hypodermic or intravenous administration of adrenalin and digitalis. Should the patient survive the algid stage, he must be fed carefully with bland liquid foods, small amounts at stated intervals, and every precaution must be observed not to provoke diarrhœa.

PLAGUE.

(Bubonic Plague; Bubonic Pest; Oriental Plague.)

Plague is an acute, specific, infectious, and slightly contagious disease caused by Bacillus pestis (Kitasato), and characterized by fever, inflammation of the lymph nodes (buboes), of the lungs, and of the cellulo-cutaneous tissues, and a general bacteremia.

Etiology.—The plague has prevailed epidemically since the early days of the Christian era, and has been answerable for great loss of life—notably during the fourteenth century, when a fourth of the population is said to have succumbed, and in 1665, when 70,000 persons died in London. Following a period of comparative quiescence, the plague recently has appeared in different parts of the world—having been disseminated from India, where it has been endemic. It has been carried to many ports of Europe, Africa, Australia, and this country, and small, local epidemics in and about the seaports have developed; but proper sanitary measures have served to prevent serious outbreaks. Age, sex, race are without etiological significance. The disease is especially common among the poor and those living under unhygienic conditions. It prevails especially during the warm seasons, but also during the colder. One attack confers partial immunity.

The exciting cause of the disease is Bacillus pestis, which may be found in the blood, lymph nodes, secretions, and viscera of patients and of subjects dead of the disease. The disease is only slightly contagious—contagion being a factor in less than 3 per cent. of the cases (Plague Commission). The infection is spread largely through the medium of rats; an epidemic among rats commonly precedes infection in man in new localities. The infection is transmitted from rat to rat, and from rat to man, by means of rat fleas—which may be carried from place to place by human subjects on their person or in their baggage, etc. Recently in California the ground squirrel has been found to be infected, and doubtless propagates the disease. Infection is acquired in the great majority of cases by means of the bites of infected rat fleas, but it may also be acquired by direct inoculation into a wound or abrasion (handling of infected material, laboratory investigations, etc.), by inhalation (pneumonic type), and perhaps by ingestion (contaminated food and drink—probably rare).

Pathology.—The lesions vary somewhat with the type of the infection, but in all there is a general plague bacillemia; the lymph nodes are swollen, hyperemic, later hemorrhagic, purulent, and more or less diffluent; the lymphatic vessels are inflamed, and about both there is more or less œdema. Hemorrhagic foci are present in the skin, the serous membranes (pleura, pericardium, peritoneum), and in the mucous membrane of the gastro-intestinal tract. The liver and kidneys show cloudy swelling and often hemorrhages, and the spleen is much enlarged and softened. In the pneumonic type there is swelling and congestion of the bronchial mucous membrane, and foci of bronchopneumonia.

Symptoms.—The period of incubation is usually two or three days, although it may be as long as five or seven. Several varieties of the disease may be distinguished: bubonic plague, pneumonic plague, cellulocutaneous plague, septicemia plague, and pestis minor.

Bubonic or glandular plague is the most common (80 per cent. of the cases). In most cases, following several days of irregular prodromes, such as malaise, chilliness, and headache, the disease develops suddenly with fever (103° to 104° F.), increased headache, anxiety, restlessness, and general neuromuscular pains or aching. The pulse becomes rapid (a valuable diagnostic sign), the respirations increased in frequency, the face and the conjunctiva injected, the tongue coated, the bowels constipated (though diarrhœa may supervene), and the patient becomes greatly depressed. Usually there is a polynuclear leukocytosis. In severe cases the patient may become collapsed, the urine scanty, almost suppressed, delirium may supervene, and in uremic coma or convulsions the patient may die, even within the first twenty-four hours (pestis siderans). In three-fourths of the cases, however, at the end of twenty-four or forty-eight hours (rarely later), the characteristic lymph-node swellings (buboes) develop, usually on one side, sometimes on both, and most frequently in the groin, less commonly in the axilla, the neck, or internally. The swellings vary much in size,

up to that of an orange, and they are tender and painful. Soon periglandular swelling and œdema develop to such a degree as to obscure or obliterate the outlines of swollen lymph nodes. By the second or third day the fever has reached its maximum; it often drops somewhat on the third or fourth day, then rises (sometimes to 108° F.), and in favorable cases by the seventh or the eighth day begins to fall by lysis and gradually reaches the normal. The glandular swellings may undergo resolution or suppuration (commonly a good sign); in some cases, however, more or less extensive sphacelation and generalized furunculosis occur. In other cases concurrent with the development of the buboes hemorrhages into the skin occur (plague spots, "fatal tokens"—whence the designation "black death"). Hemorrhages from the mucous membranes are also encountered. In the event of suppuration, the purulent discharge may rapidly abate, or the sinuses may become indolent and the patient eventually die of exhaustion, amyloid disease, or secondary infections. The mortality is about 75 per cent.

Pneumonic plague is characterized by the development of patches of bronchopneumonia. The disease begins with the ordinary manifestations of pneumococcic infection of the lung (pneumonia), for which it is frequently mistaken. The fever is high and may become intermittent; the patient becomes delirious, prostrated, and much sicker than the physical signs in the lung indicate. The sputum is thin, serosanguineous (not rusty), and contains plague bacilli in abundance. Death occurs usually before the fifth day. The mortality is very high—96 to 100 per cent.

Cellulocutaneous plague is characterized by inflammation and necrosis of the skin and subcutaneous tissues, and is often associated with involvement of the lymph nodes. The local lesions usually spread from day to day until a very large area may become involved, whereupon in favorable cases the process may become circumscribed by a well developed line of demarcation, and in the course of time a spachelus may be discharged. The general manifestations are those mentioned under bubonic plague.

Septicemic plague is a severe plague bacteremia—the general lymphatic system and the blood being overrun with plague bacilli. The fever is usually moderate only (100° to 102° F.), the depression extreme, and hemorrhages common. The patient usually dies before the lymph nodes become palpably enlarged. The mortality is over 95 per cent.

Pestis minor, pestis ambulans, is a mild form of the disease, characterized by mild fever of several days' duration, swelling and possibly suppuration of the lymph nodes, but usually with general symptoms so slight as not to necessitate the patient's taking to bed. The patients, however, are infectious, and may give rise to widespread dispersion of the disease.

Complications and Sequels.—Complications and sequels are rather common. Suppurative disorders of the eye and of the ear (leading to loss of sight and of hearing), irritability and rapid action of the heart,

divers nervous disorders and paralyses, and general asthenia are among the more common.

Diagnosis.—During an epidemic the disease is readily recognized. In case of doubt, fluid obtained from a bubo (by a sterile hypodermic needle), the blood, or the sputum should be examined for the plague bacillus; and recourse may be had to the agglutination test. Cultures and animal inoculations should be undertaken in the suspicious cases in the absence of an epidemic.

Prognosis.—The prognosis is bad; the mortality varies with the type of the infection from 50 to 100 per cent.

Treatment.—Prophylactic measures of the strictest sort should be rigidly carried out. The patient should be isolated, all utensils and the body linen should be disinfected or destroyed, rats and similar animals should be killed and their carcasses burned (as well as dead bodies); attendants should observe all sanitary precautions (page 29), and should wear close-fitting undergarments so as to prevent as far as possible bites of fleas. Haffkine's prophylactic injections, or "vaccine," consisting of gradually increasing doses of heat-killed bacilli, seem to confer more or less immunity for from six to eighteen months, especially if a second injection is made about a month after the first. This is true also of Lustig's preventive inoculations. At all events, the measure merits continued trial.

The treatment of the developed disease is purely symptomatic: hydrotherapy for the headache, delirium, and other nervous symptoms; morphine for the neuromuscular pains and general restlessness; and stimulants (strychnine, camphor, ether, whiskey, hypodermoclysis, adrenalin chloride, etc.) for the depression and collapse. An ice-bag, or belladonna ointment, or glycerin and belladona, or evaporating lotions will diminish the pain of the buboes—which, when suppuration has occurred, should be incised. Cantlie recommends early purgation with calomel and salines, and free stimulation. The evidence for or against Yersin's and Roux's antitoxic serum is not yet conclusive, although some good results have been reported.

GLANDERS.

(*Farcy; Malleus Humidus.*)

Glanders is a specific infectious and highly contagious disease of horses caused by Bacillus mallei (Loeffler-Schutz), and communicable to man; it characterized by the development of a granuloma that ultimately undergoes softening, and by general septicemia.

Etiology.—Glanders is preëminently a disease of horses, but occurs sometimes in asses and mules, and is rarely transmitted to men. Infection is transmitted directly to men, and is acquired through the medium of a wound or abrasion of the skin or the oral or nasal mucous membrane; but the ingestion of infected meat may also transmit the disease.

Pathology.—Glanders is one of the infectious granulomas, and consists of foci of epitheloid cells, giant cells, small round cells, and the specific

bacillus; these foci tend to undergo softening and form ulcers or abscesses. Two forms are recognized: (1) Glanders, in which the lesions develop in the respiratory mucous membrane, especially the nose, and where by breaking down they result in the formation of ulcers; and (2) farcy, in which the lesions develop in the subcutaneous tissues, especially the lymphatic vessels and nodes, and where by breaking down they result in the formation of abscesses which may discharge through sinuses. Both forms are commonly associated; either may antedate the other. Furthermore, a bacteremia may ensue, and lesions may be found in any of the internal organs—the lungs (bronchopneumonia), the liver, the spleen, the kidneys, the brain, the muscles, etc.; abscesses are then common, as well as hemorrhages into the serous and the mucous membranes.

Symptoms.—The period of incubation is from three to seven days, rarely as long as three weeks. Acute and chronic forms may be distinguished.

In *acute glanders* the onset is abrupt, and is characterized by fever, chilliness, headache, neuromuscular pains in the limbs and joints, and the local lesions—swelling and congestion of the nose, which is painful and tender and discharges a sanious purulent fluid. Examination reveals considerable nodular swelling of the nasal mucous membrane, and in the course of several days more or less extensive ulceration, which may progress to necrosis of the bone. Lymphangitis is marked and the adjacent lymph nodes are swollen and tender. An eruption, at first papular, but soon becoming pustular, and often leading to ulceration, occurs on the face, about the joints, and sometimes on the trunk; it has been mistaken for smallpox. Pneumonia, arthritis, and other evidences of bacteremia usually develop, and the patient dies within ten days.

Chronic glanders resembles and is usually mistaken for chronic coryza. It is likely to escape diagnosis for some time, unless the nodular swelling and ulceration attract the attention of an observing physician. It may last for months, and the patient may eventually recover.

Acute farcy, the result of cutaneous infection, resembles ordinary pyococcic infections, or septicemia. The site of the inoculation often presents phlegmonous inflammation, the lymphatic vessels become inflamed and thickened, and along their course nodular swellings (so-called farcy buds) develop, and usually go on to suppuration. Swelling and suppuration of the lymph nodes, septic arthritis, disseminated abscesses, in short the manifestations of pyemia, ensue, and the patient usually dies within two weeks. The nose is not involved and the cutaneous rash of ordinary glanders is not present.

Chronic farcy presents the local lesions of acute farcy but with little reaction, and without the severe general manifestations. The farcy buds often soften and form abscesses, and ultimately deep, more or less indolent ulcers. The disease may go on for years, but finally death results from exhaustion or acute pyemic glanders.

Diagnosis.—Acute glanders and farcy are usually readily recognized, but the chronic forms often escape detection; in both, the occupation of the patient may afford the first clue. In suspected cases some of the discharge should be examined for Bacillus mallei. Some of it may be injected into the peritoneal cavity of a male guinea-pig, the testicles of which, in the event of glanders, at the end of two days, become swollen and soon suppurate, and in the course of two weeks the animal dies of generalized nodular glanders (Strauss' test). The mallein reaction (similar to the tuberculin reaction) is also of diagnostic value in suspected cases. In acute cases the infecting bacillus may be cultivated from the blood.

Prognosis.—The prognosis is bad.

Treatment.—Treatment in the acute cases is usually unavailing, but supportive measures should be employed. In the more chronic cases surgical measures are indicated. Potassium iodide internally and mercury by inunction are said to have been of much service in some cases. Mallein has not yet proved of much value as a therapeutic remedy. Some encouraging results following the use of bacterial vaccines have recently been reported.

ANTHRAX.

(Malignant Pustule; Anthrax, or Mycosis Intestinalis; Anthrax, or Mycosis Pulmonalis; Splenic Fever; Wool-sorter's Disease; Rag-picker's Disease; Charbon.)

Anthrax is an acute, specific, infectious disease caused by Bacillus anthracis (Pollender-Davaine), and characterized by local œdema and inflammation, and often by bacteremia.

Etiology.—Anthrax is a widespread disease of the lower animals—most common among the herbivora, less common among the omnivora, and least common among the carnivora. It frequently leads to extreme devastation of herds of cattle and of sheep, especially in Russia, Siberia, and certain parts of Europe—where it is much more frequent than in this country. Infection in animals is acquired by direct inoculation—through the bites and stings of infected insects, and through feeding upon infected grain (infected by the feces of diseased animals, for instance), and upon the carcasses of animals dead of the disease. Infection may be transmitted to man directly through the bites and stings of infected insects (flies, mosquitoes, etc.), through the secretions of infected animals, through handling infected hides, hair, or wool, or through the ingestion of infected food (milk, butter, meat, sausage, etc.). Infection is acquired through a cutaneous wound, through the respiratory tract (inhaling infected dust), or through the gastro-intestinal tract. The disease is most common among stablemen, butchers, tanners, wool-sorters, rag-pickers, hair-cleaners, brush-makers, etc. Most of the cases in this country result from handling imported hides, hair, and wool. Either the bacilli or their spores may transmit the infection. One attack of the disease confers a partial immunity.

Pathology.—The lesions in man vary, depending upon the mode of infection; cutaneous, respiratory, and gastro-intestinal forms may be recognized, but in many cases the lesions are widespread. Cutaneous infection occurs most frequently on exposed surfaces (the hand, arm, face, neck), and is characterized by the development of the so-called malignant pustule (which, however, is a misnomer, since the lesion is in no way related to ordinary pyococcic pustules). Preceded usually by itching, a papule develops at the site of inoculation; this soon becomes a vesicle, filled with a clear, more commonly a sanious, fluid, and surrounded by a wide area of inflammatory œdema and induration. By the end of thirty-six hours the vesicle has dried and a blackish eschar has formed; about this there may be a series of vesicles. The inflammatory phenomena spread along the lymphatics and give rise to lymphangitis, lymphadenitis (and sometimes phlebitis), and to the most extreme and widespread œdema. I have seen a small lesion of the chin give rise to extreme œdema of the face, neck, arm, and chest. Respiratory infection results in severe bronchitis, bronchopneumonia, pleuritis, swelling of the bronchial lymph nodes, and marked œdema of the mediastinal tissues and sometimes of the neck. Intestinal infection results in the development in the mucosa of the small intestine, the stomach, rarely the large intestine, of small foci of inflammatory œdema and hemorrhage progressing to central necroses, and swelling of the mesenteric lymph nodes. Secondary intestinal infection is common in the other forms of the disease. In most cases bacteremia develops at the end of forty-eight hours. In fatal cases the spleen is enlarged, softened, and hemorrhagic, and the ordinary manifestations of toxemia (parenchymatous degeneration, etc.), as well as small hemorrhages, are encountered in the different organs.

Symptoms.—The period of incubation in cutaneous infections is from three to six days, at the end of which time, attended by chill or chilliness, fever (102° to 103° F.), headache, malaise, the above-described "malignant pustule" develops. Despite the extensive swelling, pain is rarely a conspicuous symptom. The fever often lasts only two or three days, after which the temperature may become normal. In unfavorable cases symptoms of intestinal infection (vomiting, diarrhœa, etc.), and cerebral symptoms, such as delirium, and coma, develop, the pulse becomes rapid and weak, profuse sweats ensue, and the patient may die in less than a week. In more favorable cases the eschar is separated and cicatrization proceeds, the swelling subsides, and the patient eventually recovers. All of the three patients that I have seen recovered.

Malignant anthrax œdema is the name given to a condition differing from the foregoing in the absence of a local lesion. The œdema is secondary to the general infection, involves the face, head, and sometimes the arms, and is often so extreme that gangrene results, and the patient usually dies.

In *pulmonary anthrax* the usual manifestations of severe bronchitis and of bronchopneumonia develop—chill, high fever (103° F.), cyanosis,

cough, expectoration (often hemorrhagic and containing the anthrax bacillus), prostration, profuse sweats, and collapse. Crackling rales and harsh bronchovesicular breathing, sometimes scattered foci of dulness (due sometimes to patches of solidification and sometimes to enlarged lymph nodes), and bronchial breathing may be detected. So severe is the infection in some cases that the patient succumbs within twenty-four to forty-eight hours under manifestations of profound intoxication.

In *intestinal anthrax* the symptoms usually resemble severe poisonings —sudden onset, with chill or chilliness, fever, nausea, vomiting, headache, pains in the back and legs; later diarrhœa, hematemesis, and sometimes blood (and the anthrax bacillus) in the stools, cutaneous and mucous membrane petechiæ, and symptoms of toxemia, such as coma, etc., may ensue. Death usually results in from two to five days. Intestinal symptoms are common in all forms of the infection.

Coma and delirium are not infrequent in all forms of the infection, and are attributable in most cases to toxemia, but in some fatal cases an unusual infection of the cerebral vessels with the anthrax bacillus has been encountered. On the other hand, in quite a few cases the mind is clear through the disease.

Diagnosis.—The cutaneous lesions are quite characteristic and scarcely to be mistaken for anything else. From the ordinary carbuncle they may be distinguished by the absence of pus and of the slough (so-called core). The diagnosis may be confirmed by examining some of the fluid for the anthrax bacillus, or injecting it into a guinea-pig or a mouse, which, in the event of anthrax, succumbs in a few days to anthrax bacteremia. The occupation of the patient may afford the first clue as to the real nature of the disease in internal infections (respiratory and intestinal).

Prognosis.—The prognosis depends upon the severity of the infection. The mortality in cutaneous infections about the face is about 25 per cent.; in those about the hand, 10 per cent.; in internal infections, about 90 per cent.

Treatment.—Preventive inoculation of animals (Pasteur) with an organism of reduced virulence has been practised in certain parts of the world with asserted good results. Serum therapy as yet affords us no specific in the treatment of the developed disease, although some good results have followed the use of Sclavo's serum. In cutaneous infections the local lesion should be destroyed with the cautery or excised; the wound then should be cauterized with carbolic acid, and treated with antiseptic dressings (carbolic acid and mercuric bichloride.) Injections into the advancing œdema of carbolic acid (2 per cent.) or of mercuric bichloride (1 to 4000) may be of service. Large doses of quinine internally have seemed of value in some cases. Stimulants and good food are required in all cases.

MALTA FEVER.

(Mediterranean Fever; Gibraltar Fever; Rock Fever; Neapolitan Fever; Undulant Fever.)

Malta fever is a subacute or chronic, specific, infectious, and non-contagious disease caused by Micrococcus melitensis (Bruce), and characterized by peculiar undulatory febrile attacks, profuse sweats, swelling of the joints, enlargement of the spleen, and a specific serum reaction.

Etiology.—Malta fever is endemic in the Island of Malta, and in the other islands and along the shores of the Mediterranean Sea; and it has been observed in China, India, Porto Rico, the Philippines, and the United States (imported cases). It is not unlikely that certain of the ill-understood fevers of the tropics, especially of Central and South America, may ultimately be determined to be of similar nature. The disease occurs at all periods of life, but is especially frequent between the sixth and the thirtieth year. Sex is devoid of etiological influence. The development of the disease seems to be favored by dry weather, whence it is most common during June, July, and August.

The specific cause of the disease is Micrococcus melitensis. In Malta the British Army surgeons seem definitely to have proved that the infection is derived from the goat; 10 to 15 per cent. of these animals have the micrococcus in their milk, and their blood serum gives a positive agglutination test. With this infected milk the disease was transmitted to monkeys; and stopping the use of the milk remarkably reduced the disease among the British troops. Whether the disease may be otherwise spread, for instance, by other food or drink, by the air, by the bites of insects (mosquitoes, as has been suggested), by fomites, etc., and whether it may be acquired by inhalation and direct inoculation (or through the conjunctiva, as in Strong's case), as well as by ingestion, have not yet been definitely determined. One attack of the disease confers a more or less permanent immunity.

Pathology.—The conspicuous lesions consist of enlargement of the spleen, which is hyperemic, soft, and almost, if not quite, diffluent, and contains an excess of lymphoid cells; hyperemia of the gastro-intestinal tract (occasionally progressing to inflammation and ulceration), of the liver, of the kidneys (sometimes progressing to glomerulonephritis), and sometimes of the meninges. The spleen contains large numbers of the specific micrococcus, which may be cultivated without difficulty.

Symptoms.—The period of incubation varies from three to twenty days. The onset is gradual and marked by lassitude, malaise, headache, neuromuscular pains in the limbs, anorexia, and fever, which increases day by day, in a more or less well-marked "step-ladder" fashion, until at the end of a week or ten days it reaches 103° or

104° F., in the evening. The morning remissions of the fever are often attended by profuse perspiration. As the disease progresses the headache may become severe; the liver and spleen are found enlarged and tender; symptoms of bronchial and of intestinal catarrh may supervene (though constipation rather than diarrhœa is the rule); divers joints may become swollen, tender, and painful; and the patient usually emaciates. At the end of three weeks (or sooner) the fever gradually subsides, the other symptoms abate, and the patient apparently enters upon a convalescence. At the end of two or three days of apyrexia, however, the fever and the other manifestations gradually return, and the patient is in a relapse. A series of such relapses, with pyrexial periods varying from one to three weeks and separated by periods of apyrexia of from one to three days duration, may prolong the disease into months —even a year or more. The long-continued fever, often with remissions so great as to simulate malaria, and the profuse sweats, lead usually to marked loss of strength and flesh.

As a rule, in from seventy to ninety days, convalescence ensues, but it may be unusually slow and protracted (six to twelve months), and is not infrequently complicated by arthritis, inflammation of fibrous structures, neuralgias, orchitis, etc. Death may occur, in scarcely more than 2 per cent. of the cases, from exhaustion or an intercurrent disorder.

Several types of the disease are recognized. The term undulatory is usually applied to the common form. Occasionally the onset is rather sudden, and death may occur within ten days—acute or malignant type. In other cases the constitutional symptoms are slight, consisting only of slight evening fever, malaise, and progressive emaciation, though more severe symptoms may develop at any time—so-called intermittent type.

Diagnosis.—Malta fever must be distinguished from typhoid fever and from malaria. Malaria is readily excluded by the absence of the malarial parasite from the blood and the non-response to quinine. Most cases, at least in regions remote from the Mediterranean, are mistaken at the beginning for typhoid fever, but the absence of many of the characteristic symptoms of typhoid fever should be deemed significant. The diagnosis is rendered certain by the absence of the Gruber-Widal serum reaction with Bacillus typhosus and its presence with Micrococcus melitensis (in some cases as early as the fourth day of the fever). The peculiar character of the fever also is quite characteristic, even in the absence of the specific serum reaction. The micrococcus in some cases may be cultivated from the blood.

Prognosis.—The duration of the disease is long, but the mortality is low—not more than 2 per cent.

Treatment.—Treatment is purely symptomatic, since an efficient serum- or vaccine-therapy has not yet been produced. Hydrotherapy and the general measures suitable in typhoid fever are indicated.

During the periods of pyrexia it is wise to adhere to the diet recommended in typhoid fever. Drugs, so far as known, seem to have no influence on the course of the disease. Convalescence is promoted by tonics, change of climate, or a sea voyage.

LEPROSY.

Leprosy is a chronic, specific, infectious disease caused by Bacillus lepræ (Hansen), and characterized by granulomas involving the skin, the mucous membranes, the nerves, the lymphatics, and the viscera.

Etiology.—Leprosy is known to have prevailed centuries before the Christian era. At present it is found in all parts of the world, but is endemic in India, China, Japan, and other parts of Asia, Africa, Russia, Persia, Norway, Sweden, Spain, Portugal, Central and South America, the West Indies, Mexico, Iceland, Canada, Australia, the Sandwich Islands, etc., and in our own United States, especially Louisiana and other southwestern States. From time to time isolated cases have been observed in many, if not all, the States, but most of the cases have been imported.

The exciting cause of the disease is Bacillus lepræ, which is found in leprous lesions, in the secretions therefrom, and in the saliva, and oral and nasal discharges in the event of lesions about the nose and mouth. The disease is not markedly contagious, since prolonged and rather intimate personal contact seems necessary for infection; but that the disease may be spread by fomites cannot be denied. It has been suggested that fleas, lice, bed-bugs, and the itch parasite are the active agents in transmitting the infection. Hutchinson believes that infection is spread largely by uncooked fish, in confirmation of which opinion he points to the prevalence of the disease in fish-eating communities. How infection is acquired is not definitely known: should we believe Hutchinson, it may be acquired by ingestion; it may be acquired by inhalation—witness the lesions in the nose, believed by some observers to be an initial lesion; and the frequency of leprosy of the hands and the feet (often uncovered), together with the presence of lepra bacilli in the earth, suggests that infection may be acquired by direct inoculation through wounds. Heredity is of little, if any, etiological influence; the youngest subjects are rarely less than three to six years of age; beyond this no age is exempt.

Pathology.—Leprosy is one of the so-called granulomas. The lesions consist of nodular masses or diffuse infiltrations made up of small rounded and fusiform cells, larger endothelial often vacuolated cells, and giant cells, in and between which the lepra bacilli are found in profusion. These lesions are found usually in the skin, the mucous membranes, and the viscera. On the surface the lesions not infrequently progress to softening and ulceration attended by marked loss

of tissue and noteworthy mutilation. In the nerves nodular and diffuse infiltrations are observed, with overgrowth of the connective tissue and atrophy of the axons. Changes analogous to tabetic arthropathy are sometimes observed in the bones. The lymph nodes are often palpably enlarged.

Symptoms.—The period of incubation is from three to five years (Hansen), but a case of presumed direct inoculation has been observed in which the period of incubation was four weeks. Some observers believe in the existence of a primary sore (the existence of which is not altogether unlikely), and have located it commonly in the nose (where they have observed ulceration), but the evidence is not yet conclusive. During the period of incubation the patient suffers from irregular attacks of fever, malaise, headache, and pains in the joints. In many cases the initial manifestations consist of dusky red erythematous and hyperesthetic patches on the skin, especially of the face, extensor surfaces of the hands and forearms (macular leprosy); these soon become pigmented, later anesthetic, and gradually lose their pigment and become white (lepra alba). Soon the disease takes on the characters of one of the two well-recognized types, the nodular (tubercular) or anesthetic; but commonly the disease is of mixed type.

The *nodular type* is characterized by the development of nodules, varying in size from that of a pea to that of a cherry, and of diffuse fibrous infiltration of the skin and the mucous membranes. Beginning usually in the skin of the face (nose, lips, or chin) or the ears, they produce painless, irregular thickenings, nodosities, and folds, leading to much distortion and ultimately to the condition known as facies leontina. Gradually nodules and thickenings develop elsewhere, especially on the hands, forearms, feet, legs, buttocks, knees, etc., until much of the cutaneous surface is involved. Occasionally some of the nodules lessen in size, and even disappear, but usually they soften and ulcerate, and thus add to the already existing deformity. Involvement of the mucous membrane of the nose, mouth, throat, and larynx is evident in the nasal discharge, hoarseness, aphonia, dyspnœa, etc., and is quite manifest upon local examination—irregular nodular and diffuse thickenings and ulcerations. Inhalation pneumonia not infrequently results; and from involvement of the conjunctiva and cornea blindness may ensue. The disease runs a course of years (five to ten or more), and ultimately leads to death from exhaustion or from pulmonary or other complications.

The *anesthetic type* manifests itself, in addition to the macules already mentioned, by early pain in the limbs and areas of hyperesthesia and paresthesia, followed by anesthesia (often only analgesia) and trophic lesions (bullæ) of the skin of the extremities. Anesthetic areas, however, may develop without preceding macules, and not infrequently become extensive. The bullæ often break down and lead to the formation of extensive perforating ulcers and widespread destruction of tissues. The nerve trunks are often palpably thickened; the muscles waste;

the fingers become clubbed and contracted (claw-like); wasting, necrosis, and gangrene ensue; and the fingers and toes may drop off (leprosy mutilans); or arthropathies (resembling those of tabes) may develop. The lesions pursue an extremely chronic course (ten to thirty years), and ultimately lead to death through exhaustion or an intercurrent infection.

Diagnosis.—In advanced cases the diagnosis is unmistakable. In the early stages the dusky red areas of erythema, at first hyperesthetic, later anesthetic, and devoid of pigment; the nodular neuritis; and the detection of lepra bacilli in the nasal secretion or in an excised nodule, suffice for diagnosis. Serum diagnosis—the agglutination of the lepra bacilli by the blood serum of a leprous patient—may prove of some service.

Prognosis.—The patients frequently live from ten to thirty years, unless carried off by an intercurrent infection. In some cases the disease seems to have been entirely arrested.

Treatment.—Segregation is advisable in all cases. Special attention should be paid to general hygienic measures, and the patient should have an abundance of good nutritious food, of sunshine, and of fresh air. Recently almost specific properties have been claimed for chaulmoogra oil, 10 minims, gradually increased to 2 drams, three times daily. Dyer reports several cures from its use. Dyer has also had good results from Calmette's antivenene. Gurjun oil, ichthyol (Unna), mercuric bichloride, salicylic acid, and many other remedies have been recommended from time to time. A supposed antileprous serum also has been introduced of late. Nastine, a bacterial fat recommended by Deycke, does not seem to be of much value.

NON-BACTERIAL FUNGUS INFECTIONS.

The non-bacterial fungus infections comprise a group of diseases caused by microörganisms that are sometimes described as higher bacteria, sometimes as fungi, but which probably represent low forms of life intermediate between the bacteria and the more highly developed fungi. They have been incompletely studied and classified, but Park suggests the following attempt at differentiation: (1) Actinomyces, characterized by radiating wreath-like forms which it alone produces in the living body. (2) Streptothrix, characterized by an abundant true branching, wavy growth, later fragmentation, and the formation of conidiæ, which serve as organs of propagation, and in this sense may be considered as spores. (3) Cladothrix, characterized by false branching, rapid fragmentation, and then bacillary characteristics in old cultures. (4) Leptothrix, characterized by lack of observed branching, non-wavy growth, but, on the contrary, stiff, almost straight threads, in which division processes are seldom or never observed.

ACTINOMYCOSIS.

Actinomycosis is a chronic, specific, infectious disease caused by Streptothrix actinomyces, or ray fungus, and characterized by the development of a chronic granuloma that ultimately softens and discharges characteristic actinomycotic granules.

Etiology.—Actinomycosis is common in cattle, in whom it causes "big jaw" and "wooden tongue," in horses, and in pigs. It is believed to be non-contagious, since direct transference from the sick to the well is not observed. Inasmuch as the fungus occurs on grain, such as oats, barley, rye, etc., infection is probably acquired with the food—certainly in the lower animals. In man infection may be acquired in a similar manner, an abrasion of the mucous membrane or a carious tooth (in which the fungus has been found) being probably the infectious atrium; but it may be acquired also by inhalation (as at threshing, for instance), or through a cutaneous wound. The disease is three times as common in men as in women.

Pathology.—Actinomycosis is one of the so-called infectious granulomas, and consists of foci of epithelioid cells, giant cells, small round cells, and the fungus, and the later development of marked hyperplasia of the connective tissue—a condition not altogether unlike tuberculosis; but so great is the enlargement that it is sometimes mistaken for sarcoma. Later softening and suppuration occur and many sinuses form, from which issues a thin, sanious, purulent fluid containing characteristic, small, sulphur-yellow, actinomycotic granules which, upon microscopic examination, reveal the ray fungus.

Symptoms.—Almost any organ of the body may be involved, but for obvious reasons, the jaw and the adjacent tissues are most frequently affected. Usually a firm, indurated mass develops; this gradually increasing in size, ultimately softens, whereupon its contents are discharged through several sinuses. In consequence of involvement of the periosteum and the bone, necrosis not infrequently ensues, and gradually other organs become implicated, such as the tongue, the tissues of the neck, the ear, and sometimes even the meninges, from direct extension, and more distant organs, such as the liver, the lung, the brain (symptoms of tumor), by metastasis. The region of the cecum and appendix is not infrequently the seat of primary involvement, but any part of the large or the small intestine may be involved, and the actinomyces may be found in the stools. The liver is rather frequently the seat of secondary abscesses (reticulated or honey-combed abscesses). The lungs may reveal miliary actinomycosis, fetid bronchitis, chronic interstitial pneumonia, or bronchopneumonia, which may progress to abscess or cavity formation or gangrene; the actinomyces may be found in the sputum. The pleura may be markedly thickened, and from this by contiguity the adjacent soft parts, the ribs, or the vertebræ may

become involved. Cutaneous lesions and chronic suppurative and ulcerative processes have occasionally been observed.

Diagnosis.—The disease may be suspected in cases of long-standing suppuration with a tendency to involvement of the bones, but it is confirmed only by finding the specific cause, the actinomyces.

Prognosis.—The prognosis depends largely upon the extent of the lesions and whether or not they are amenable to surgical intervention.

Treatment.—The treatment is primarily surgical. But recently in view of the success that has attended its administration potassium iodide, upward of one dram (4 grams) daily, has come to be looked upon as almost a specific. Good results have also attended the use of the *x*-rays.

MYCETOMA.

(*Madura Foot.*)

Mycetoma is a chronic, specific, infectious disease caused by Streptothrix maduræ or Streptothrix mycetomæ, and characterized by nodular enlargement and deformity proceeding to oily degeneration and sinus formation.

Etiology.—Mycetoma has been observed, usually sporadically, in Asia, Europe, South America, and the United States. It is most common between the twentieth and the forty-fifth year, and occurs more frequently in men (especially those who go bare-footed) than in women. How infection occurs is not definitely known, though it is not unlikely through a wound or abrasion, or, as some believe, through the sweat glands.

Symptoms.—Two varieties of mycetoma are recognized, the melanoid, or mycetoma with black granules, and the ochroid, or mycetoma with yellowish or white granules—due respectively to Streptothrix maduræ and Streptothrix mycetomæ. The disease begins with the development of a hard, firm, painless nodule on the sole of one foot, usually the right, though other parts may be affected, such as a toe, the hand, or a finger. The nodule soon softens and at the end of about a month ruptures and discharges a peculiar, viscid, oily, slightly purulent, and sometimes blood-streaked fluid in which the blackish or yellowish granules are suspended. Similar lesions develop elsewhere in the foot, which ultimately becomes much enlarged. The lesions gradually attack the muscles, ligaments, and bones and convert them into a thick, greasy, yellowish mass into which a probe may be inserted in any direction to a considerable depth without provoking pain or hemorrhage. The lesions show little or no tendency to heal, the sinuses being, as a rule, permanent. As the foot enlarges and can no longer be used, the leg atrophies from disuse. In some cases the lesions tend to spread along the lymphatics and set up new foci of infection. The general health remains unimpaired for a long time; the patient may live ten or twenty years, but ultimately succumbs to exhaustion or an intercurrent infection.

Diagnosis.—The disease is only likely to be mistaken for actinomycosis to which the disease is much allied. Doubtless many of the cases

reported, especally in this country, were really actinomycotic. The differential diagnosis is to be made largely by a microscopic examination of the discharge. In some cases the lesions may simulate those of syphilis or sarcoma.

Treatment.—The treatment consists of resection if a small area only is involved, and of amputation if the disease is more widespread. The amputation must be done well above the infected area. Potassium iodide is useless.

NOCARDIOSIS.

(*Streptothricosis; Cladothricosis; Pseudotuberculosis; Atypical Actinomycosis.*)

Nocardiosis is a generic term, suggested by Wright, to describe a group of diseases caused by nocardia, microörganisms to which the terms streptothrix, cladothrix, pseudotubercle bacillus, and atypical actinomycosis have also been applied.

Etiology.—These nocardia are widely distributed in nature. Infection is usually acquired through inhalation, rarely by means of cutaneous or mucous membrane wounds; and it is predisposed to by general malnutrition. The infection is rare in man.

Pathology.—The lesions consist of inflammation, necrosis, suppuration, abscess formation, and granulation tissue. In some cases the lesions resemble tuberculosis, that is, there are tubercles, fibrocaseous nodules, and calcification. The lesions are most common in the lungs; metastases have been observed in the brain, kidney, myocardium, lymph nodes, etc.

Symptoms.—The process may be acute or chronic. The symptoms vary, depending upon the seat of the lesions, but in general they are those of inflammation and suppuration. When occurring, as is common, in the lung, the symptoms are those of pneumonia, abscess, tuberculosis, or empyema. In the brain, the symptoms are those of abscess, tumor, or meningitis. Multiple abscesses may occur in the skin and subcutaneous tissues.

Diagnosis.—Occasionally the symptoms have been somewhat atypical of the disease suspected, but, as a rule, the diagnosis depends solely upon detecting the causative microörganism in the sputum or other discharge or fluid of the body.

Prognosis.—The outlook is bad, since the great majority of the subjects finally succumb.

Treatment.—There is no specific treatment. In general, the treatment is that of the disease suspected—pneumonia, tuberculosis, abscess of the lung, etc. Abscesses should be treated surgically.

PULMONARY ASPERGILLOSIS.

Pulmonary aspergillosis is a chronic disease caused by Aspergillus fumigatus and characterized by inflammation and necrosis of the lung.

Etiology.—Aspergillus fumigatus is widely distributed in nature, and is often found as a harmless parasite on different surfaces and in certain cavities of the body. Most of the cases of infection occurring in man are secondary or terminal, developing especially in tuberculosis; but apparently primary cases have been observed. The infection is believed to be acquired from infected birds or infected seed fed to birds (occurring thus in pigeon feeders), or from the dust of rye flour used to remove grease from hair (whence the infection occurs in hair-sorters).

Pathology.—The lesions consist of inflammatory processes akin to those of bronchopneumonia, in the midst of which the fungus may be encountered. From coalescence of adjacent foci more or less widespread solidification may occur. Ultimately necrosis and softening may ensue, cavity formation develop, and gangrene may supervene.

Symptoms.—The symptoms are those of chronic tuberculosis, chronic interstitial pneumonia, bronchiectasis, etc., and aside from the presence of the fungus in the expectoration may not present special characteristics.

Diagnosis.—The diagnosis depends upon detecting the fungus in the sputum. In most cases, cases of secondary or terminal infection, the infection may be of minor importance; the tubercle bacillus may be found associated with the fungus.

Prognosis.—The outlook is bad, largely because of the associated conditions.

Treatment.—The treatment is that of the underlying disease, such as tuberculosis, etc. Arsenic and potassium iodide have also been recommended. Prophylactic measures, looking to the avoiding of inhaling infected, dust-laden atmosphere, should be undertaken with a view to prevent the primary cases.

OÏDIOMYCOSIS.

(Blastomycosis; Saccharomycosis; Blastomycetic Dermatitis; Protozoic Dermatitis; Dermatitis Coccidioïdes; Coccidioïdal Granuloma; Psorospermiasis.)

Oïdiomycosis is a chronic infectious disease of the skin and internal organs caused by fungi of the genus Oïdium, and characterized by granulomatous inflammation proceeding to suppuration.

Etiology.—The organism is widely distributed in nature, but the factors leading to infection in man are not well understood.

Pathology.—The lesions may be limited to the skin or the lungs, or they may involve also other organs, such as the liver, spleen, kidneys, adrenals, lymph nodes, meninges, etc. In general they consist of granulomatous formations tending to softening and abscess formation. In some cases the slow progression of the lesions and the absence of softening suggest tuberculosis. In the skin the lesions may simulate those of tuberculosis or squamous epithelioma.

Symptoms.—In the skin the symptoms consist of the development of a papular or pustular area, which gradually increases in size to form a

rough, scaly, elevated fungus-like surface, variously the seat of minute abscesses and of scars in different stages of cicatrization. In the lungs and other viscera the symptoms are analogous to those of tuberculosis.

Diagnosis.—The diagnosis depends upon the detection of the causative microörganism.

Prognosis.—The disease is chronic, lasting many years. When limited to the skin the process may be brought to a standstill or cured; but when the viscera become involved death usually eventually ensues.

Treatment.—Potassium iodide in large doses (150 grains, 10 grams, daily) has been followed by good results in some cases. The *x*-rays are also of service in some cutaneous lesions; or these lesions may be curetted, excised, or treated with antiseptics.

ZOÖPARASITIC INFECTIONS.

PROTOZOAN INFECTIONS.

The protozoa are unicellular animal organisms that reproduce by division or sporulation. There are four important phyla (tribes) or primary divisions:

1. Sarcodina, protozoa that throw out changeable protoplasmic processes, called pseudopodia, which serve the double purpose of nutrition and locomotion. To these belong the class *Rhizopoda* (those with lobose or reticulose pseudopodia). These include: Entamœba dysenteriæ (or histolytica), the cause of one form of dysentery; Entamœba coli, believed to be a harmless intestinal parasite; and Leydenia gemmipara, which has been found in ascitic fluid in cases of carcinomatosis, but is perhaps of no pathogenic significance.

2. Mastigophora, protozoa provided with undulating membranes or flagella that serve as means of locomotion and obtaining food. To these belong the class *Flagellata* (small forms with one or several flagella). These include: (*a*) Spirochætæ (spiral in shape and provided with an undulating membrane, but no flagella), of which Spirochæta recurrentis (or obermeieri) is the cause of relapsing fever of Europe, and is perhaps related to Spirochæta carteri (relapsing fever of India), Spirochæta novyi (relapsing fever of America), and Spirochæta duttoni (relapsing fever of Africa); Spirochæta vincenti is found, associated with a fusiform bacillus, in some cases of inflammation of the throat, called Vincent's angina; and Spirochæta refringens is found in ulcerations of the genitalia, and is often associated with Treponema pallidum in syphilitic lesions. (*b*) Treponemæ, those with a flagellum at both ends, but without an undulating membrane. Of these, Treponema pallidum is the cause of syphilis; and Treponema pertenuis is believed to be the cause of yaws

(frambœsia). (*c*) Trypanosomæ, those with a more or less spiral-shaped body, along one side of which there is an undulating membrane from which a flagellum projects. Of these, Trypanosoma gambiense is the cause of human trypanosomiasis, or sleeping sickness. The following trypanosomes also are found in mammals: Trypanosoma lewisi, which infects rats, but seems usually not to occasion noteworthy symptoms; Trypanosoma evansi, the cause of surra in horses and mules, especially in India and the Philippines; Trypanosoma brucei, the cause of nagana in horses, and found also in many game animals in Africa; Trypanosoma equiperdum, transmitted by coitus, the cause of dourine (*maladie du coit*) in horses and donkeys; Trypanosoma equinum, the cause of *mal de caderas* in horses in South America; Trypanosoma theileri, the cause of galziekte (gall sickness) in cattle; and Trypanosoma dimorphon, the cause of Gambian horse disease. (*d*) Leishmania (oval intracellular parasites), of which Leishmania donovani is the cause of kala-azar, and Leishmania tropica is the cause of oriental sore. (*e*) Trichomonas, a parasite with a fusiform body, an undulating membrane and three flagella arising from its anterior end. These include: Trichomonas vaginalis, which is common in acid vaginal mucus; and Trichomonas (or Cercomonas) hominis, which is found in the intestine, where apparently it may set up an acute enteritis; and it may invade or be introduced into the bladder and set up cystitis. (*f*) Lamblia intestinalis (a pear-shaped, flagellated organism), which is common in the intestine, where it may perhaps set up acute or chronic diarrhœa; it has been found also in the expectoration of bronchiectasis and gangrene of the lung, in pleural exudates, etc.

3. Infusoria, protozoa with cilia. To these belong the ciliata (those with contractile vacuoles and many fine cilia which are shorter than flagella and exhibit a sweeping motion). These include Balantidium coli, a parasite that is common in hogs, and may set up a dysenteric disorder in man.

4. Sporozoa, protozoa without motile organs, that reproduce by sporulation. These include: (*a*) *Coccidiaria*, of which Eimeria stiedæ (Coccidium cuniculi or oviforme) is most frequently found in the epithelium of the bile ducts of animals, especially rabbits, and the oöcyst of which may be found in the feces. The disorder is known as coccidiosis; several cases have been reported in man. Isospora bigemina (Coccidium bigeminum) is common in the intestine of dogs and cats, and has been observed in the human subject. (*b*) *Hæmosporidia*, of which Plasmodium malariæ is the cause of malarial fever. Similar organisms have been found in other animals: Plasmodium kochi in monkeys; Plasmodium præcox in birds (causing proteosoma; halteridium in crows and pigeons); and other hæmosporidia in other animals.

AMŒBIASIS.

(*Amœbic Dysentery; Amœbic Colitis.*)

The common manifestation of amœbiasis is an acute or chronic inflammation and ulceration of the colon caused by Entamœba dysenteriæ (or histolytica). Schaudinn, Craig, and others distinguish two forms of amœbæ that may be found in the intestine, the pathogenic Entamœba dysenteriæ, and Entamœba coli, believed to be a harmless, non-pathogenic form.

Etiology.—Amœbic dysentery is endemic in the tropics, where it is a very common, if not the commonest form of dysentery encountered. Sporadic cases occur from time to time throughout the subtropical and the temperate zones, and epidemics are by no means unknown. The disease is most common in young male adults; it prevails especially among the poor, and it is predisposed to by general malnutrition and unhygienic surroundings. The infection is usually transmitted by the drinking water.

Pathology.—The lesions may involve the entire colon, and sometimes even the lower part of the ileum; but they are most common and, as a rule, most marked in the cecum, in the hepatic and sigmoid flexures, and in the rectum. They consist of inflammation, œdema, and cellular infiltration of the submucosa, which are soon followed by necrosis and ulceration. In the early stages there are localized areas of thickening and infiltration, the mucous covering of which soon becomes soft and necrotic, and eventually being discharged, leaves a roundish, ovoid, or irregular ulcer. The base of the ulcer is often much larger than the point of discharge of the necrotic tissue, that is, the edges of the ulcer are undermined or overhanging; and adjacent ulcers often communicate by means of sinuous tracts, the mucosa overlying which may be more or less normal, although it is usually more or less thickened and inflamed. The floor of the ulcer may be made up of the submucosa, the muscularis, or the serosa; or it may be adhesions that bind the colon to adjacent organs or tissues. Perforation may occur into these adhesions or into the surrounding connective tissue or the peritoneum. In advanced cases, the intestinal wall is much thickened and infiltrated, and the seat of numerous and extensive ulcerations, with here and there areas of more or less intact mucosa; or there may be widespread sloughing, pseudomembrane formation, or gangrene. Healing of the ulcerations and cicatrization occur irregularly and to a varying degree, and they lead to more or less deformity and partial obstruction (stricture) of the bowel. Microscopic examination reveals the ordinary changes of inflammation and ulceration, with, however, only moderate polynuclear leukocytic infiltration: necrosis rather than suppuration being conspicuous. In chronic cases there is also considerable connective tissue proliferation. The amœbæ may be found in the base of the ulcer, in the adjacent lymph spaces, and sometimes in the bloodvessels—whence they are

readily conveyed to the liver, where they give rise to widespread necrosis and a so-called amœbic abscess. The amœbæ may be cultivated, with difficulty, from the intestinal discharges, the intestinal wall, and the liver abscesses. In the intestinal tract they may be associated with other protozoa, such as trichomonas, cercomonas, etc., and with various metazoa, such as Tænia saginata, Ascaris lumbricoides, Uncinaria duodenalis, etc. They have been found also in the peritoneal cavity, the pleura, lung, bladder and urine, abscess of the mouth, necrosis of the jaw, etc.

Symptoms.—Strong divides the cases into three classes, which, however, are not sharply demarcated the one from the other: (1) Mild cases and those of moderate severity; (2) cases with acute onset; and (3) advanced and chronic cases. In the mild cases the onset is insidious, the patient often being unable to state when the disease began. The chief symptoms consist of more or less abdominal pain and diarrhœa; the diarrhœa may be periodic and may alternate with constipation. The stools may contain mucus and the amœbæ; often little if any blood. In addition there may be such general symptoms as lassitude, headache, loss of weight, etc. The disorder may last for months, the mildness of the symptoms often warranting the terms latent or masked infection; but the disorder may become aggravated and pass on to one of the other types. The cases with acute onset may represent an acute infection or an acute exacerbation of a mild or latent infection. The chief symptoms consist of abdominal pain (tormina), frequent stools containing mucus, blood, and the amœbæ, and more or less constant and distressing tenesmus. Sloughs of the bowel also may be passed. Usually there is abdominal tenderness, especially along the course of the colon, moderate fever, and the patient emaciates rapidly. The pulse may become rapid and feeble, and death may ensue within a week or ten days from asthenia, toxemia, syncope, hemorrhage, or peritonitis from perforation. In other cases, after four or five days, the acute manifestations subside and may eventually disappear, or the condition may become chronic. The advanced and chronic cases may result from the acute cases or they may be subacute or chronic from the beginning. They are characterized by alternating periods of diarrhœa and constipation; the diarrhœic attacks are characterized by abdominal pain, frequent stools containing mucus, blood, and the amœbæ, more or less tenesmus, and slight or moderate fever. There may be more or less emaciation and impairment of the general health; so-called indigestion, and provoking of the attacks apparently by dietetic indiscretions. The tongue is likely to be red and raw-looking. The condition may last for six months, or a year or more. In the protracted cases emaciation may be extreme, and death may eventually ensue.

Complications and Sequels.—Liver abscess is common; it may involve the lung and lead to a so-called hematopulmonary abscess. Intestinal perforation and peritonitis, arthritis, etc., also occur.

Diagnosis.—The diagnosis is readily made from attention to the bloody mucous character of the stools, and the detection therein of the amœbæ. The liver should always be carefully studied for signs of abscess (enlargement, local pain or discomfort, chills, fever, sweats, and leukocytosis).

Prognosis.—The mild cases usually yield to treatment, but relapses are common. In chronic cases the outlook is not encouraging, but with care and attention recovery may be brought about.

Treatment.—The prophylactic measures mentioned on page 29 should be carried out, since there is much reason to believe that the disease is spread and acquired as is typhoid fever. During the acute stage the patient should be in bed. The diet should be the blandest—milk (peptonized or diluted with barley water, lime water, or Vichy water), albumin water, raw eggs, beef juice, broths, custard, milk toast, etc. In subsiding or chronic infections the greatest attention also must be paid to the diet, since recurrences are not infrequently provoked by dietetic indiscretions. At the beginning of an attack, especially if there has been little diarrhœa, the bowels should be cleared with blue mass, calomel, or castor oil to which 20 minims (1.25 c.c.) of tincture of opium may be added. Ipecac is believed by men of large experience to exert an almost specific effect, and assuredly should be tried. Not less than three hours after the patient has had food, mild counterirritation (a mustard plaster or tincture of iodine) should be applied to the abdomen, and 10 to 15 minims (0.6 to 1 c.c.) of tincture of opium given by the mouth, or $\frac{1}{4}$ grain (0.015 gram) of morphine administered hypodermicly. After the lapse of ten to fifteen minutes, 30 to 60 grains (2 to 4 grams) of freshly powdered ipecac root should be administered, preferably in salol-coated pills or capsules. The patient must maintain the recumbent posture for several hours: this as well as the salol-coated pills or capsules tend to obviate nausea and vomiting; but should vomiting occur, the dose of ipecac should be repeated after a short interval. Decreasing doses (5 to 10 grains, 0.3 to 0.6 gram, less than the initial dose) should be given on succeeding days. The use of purgative doses of sodium or magnesium sulphate has also been much commended: one dram (4 grams) at frequent intervals (every fifteen to thirty minutes) until the intestine has been thoroughly cleansed, whereupon aromatic sulphuric acid (believed to be detrimental to the infecting microörganisms) may be given in rather large doses. Irrigation of the bowel is of great advantage in many cases, but the pain and tenesmus are often obstacles to its use in acute cases; but sometimes these may be lessened by a preliminary enema of tincture of opium ($\frac{1}{2}$ to 1 dram, 2 to 4 c.c.) and thin starch water (2 to 4 ounces, 60 to 120 c.c.). The irrigation should be done by means of a fountain syringe, and the tube should be inserted high up; the best solution is quinine, 1 to 5000 gradually increased to 1 to 1000, of which one to two quarts should be injected twice daily and retained, if possible, for at least fifteen minutes. Ice-water enemas also may prove grateful. The rectal tenesmus may be relieved by an opium and belladonna suppository.

In chronic cases that do not improve on medical treatment, resort may be had to colostomy or appendicostomy and drainage and irrigation of the colon through the wound.

RELAPSING FEVER.

(Recurring Fever; Seven Day Fever; Famine Fever; African Tick Fever; Spirochetosis; Spirillosis.)

Relapsing fever, originally believed to be a single, acute, specific, infectious disease characterized by alternating periods of fever and of apyrexia of about six days each, is now known to include at least four forms of such fever, each more or less distinctive, more or less limited in geographical distribution, and caused by four species of spirochetes: Spirochæta recurrentis (Spirillum obermeieri), the cause of the relapsing fever of Europe, especially of Russia, the Balkans, etc.; Spirochæta duttoni, the cause of the relapsing fever of Africa (African tick fever); Spirochæta carteri, the cause of the relapsing fever of India; and Spirochæta novyi, the cause of the relapsing fever of America.

Etiology.—The disease prevails especially amidst destitution, squalor, and general unhygienic surroundings. Personal factors, such as age, sex, and race, as well as climate and season, are without special etiological significance, although the disease is said to prevail especially among young adults, and during the winter. One attack confers a partial immunity.

The exciting cause of the disease is one of the aforementioned spirochetes. The infection is believed to be transmitted by insects or ticks. This has been definitely proved in the case of Spirochæta duttoni, a tick, Ornithodorus moubata, being the transmitting agent. The bed-bug (Cimex lectularius) is believed to be the transmitting agent of Spirochæta recurrentis, and the body-louse (Pediculus corporis) that of Spirochæta carteri, but the evidence is not yet conclusive. Nothing is known of the mode of transmission of Spirochæta novyi. Nor has it yet been definitely determined whether the transmission is purely mechanical or whether the spirochetes undergo some developmental process within the body of the transmitting insects or ticks.

Pathology.—There are no characteristic lesions. The spleen is enlarged and soft; the liver, kidneys, heart, etc., reveal parenchymatous degeneration and, occasionally, hemorrhagic infarction; and the bone marrow has been found hyperplastic.

Symptoms.—The period of incubation is from five to eight days. Usually without prodromal symptoms, the disease begins suddenly, with severe chill, high fever, rapid pulse (120 to 140 per minute), general neuromuscular pains and sensitiveness, extreme prostration, and sometimes delirium. In children and adolescents there may be nausea and vomiting, rarely convulsions. By the end of twenty-four or thirty-six hours, the temperature has attained its maximum (105° to 106° F.),

and it continues high with slight morning remissions. During the period of fever, the spleen is much enlarged and palpable, and there may be jaundice, nausea, vomiting, slight bronchitis, profuse sweating, sudamina, herpes labialis, slight albuminuria, stupor, delirium, and a slight leukocytosis. After the lapse, usually of six (sometimes five, or seven, rarely ten) days, the temperature falls rapidly within twelve hours or less, to the normal or below, the patient sweats profusely, or has a critical diarrhœa, and by the following day feels almost entirely well, although there may be a slight elevation of temperature for a day or two. Rarely there is but a single paroxysm, but in most cases, on or about the fourteenth day, that is, after a period of apyrexia, lasting six or seven days, a severe chill occurs, and the febrile attack is repeated; sometimes there is a third, fourth, and even fifth relapse. Usually the succeeding paroxysms are of decreasing severity and duration. In unusual (toxic) cases, a typhoid condition with marked delirium and coma, sometimes petechial hemorrhages, localized necrosis of the mucous membranes, and bed sores, develop, and death occurs from toxemia and cardiac collapse.

The African tick fever resembles the other types of relapsing fever, but it may differ in some respects: the periods of fever may be as short as three days, the periods of apyrexia may be irregular (one day to three weeks), diarrhœa and dysentery may be present, etc.

Complications.—Complications are rare. The most common are pneumonia, sepsis, ititis, choroiditis, neuritis (with consequent paralysis), rupture of the spleen, gastro-intestinal catarrh, and hemorrhage from the kidneys, nose, gastro-intestinal tract, etc.

Diagnosis.—In the early stages the disease may be mistaken for typhoid or typhus, and even yellow fever, but the subsequent course of the disease and the results of examining the blood are conclusive. The spirochetes may be found in the peripheral blood during the period of fever; they lessen in number with the decline of the fever, and they are absent after the crisis and during the periods of apyrexia, although the blood remains infectious to susceptible animals. The recurrence of the paroxysms at regular intervals indicates the protozoan nature of the parasite and that it passes through a definite cycle of development. A form of lymphatic tuberculosis, clinically resembling pseudoleukemia, or Hodgkins' disease, much simulates relapsing fever, but the paroxysms of fever and of apyrexia are more irregular in course, development, and frequency; the lymph nodes may be enlarged, the spirochete is absent from the blood, and the patient may react to tuberculin injections. Irregular forms of malaria may be distinguished by the course of the fever and the results of examining the blood.

Prognosis.—The prognosis is good in the absence of extreme severity of infections and complications. The mortality rate is low—2 to 4 per cent.

Treatment.—Efforts should be made to prevent the infection by inculcating and practising habits of personal and domestic cleanliness, by avoiding vermin-infested places, and the use of beds high from the

ground and protected by mosquito netting, or of hammocks suspended by ropes. The treatment of the developed disease,which must be based upon general principles, consists largely of good hygienic surroundings, good nursing, and good food. The symptoms are to be treated symptomatically—the high fever by cold sponging, the neuromuscular pains by local applications, embrocations, and anodynes, such as Dover's powder internally or morphine subcutaneously. Toxemia and cardiac debility are to be treated on general principles. The evidence in favor of an antispirochetic serum is not yet conclusive; Novy and Knapp have had success in curing the infection in rats with an immune serum.

SYPHILIS.

(*Lues; The Pox.*)

Syphilis is a chronic, specific, infectious, congenital or acquired disease, caused by Treponema pallidum (Schaudinn and Hoffmann); it is characterized, when acquired, by a so-called primary lesion, the chancre, at the seat of inoculation; certain constitutional disturbances, and disorders of the skin, mucous membranes, and lymph nodes (so-called secondaries); specific granulomatous growths in the viscera, blood-vessels, bones, muscles, skin, and mucous membranes (so-called tertiaries); and sometimes by certain so-called parasyphilitic or metasyphilitic diseases of the nervous system, especially tabes dorsalis and general paresis (so-called quartanaries). Aside from the primary lesion, the lesions of the congenital form of the infection do not differ essentially from those of the acquired form.

Etiology.—Syphilis prevails in all parts of the world; it is peculiarly a disease of mankind, and occurs at all ages and in both sexes. Until recently the lower animals were believed to be immune, but Metchnikoff and Roux have shown that experimentally the infection can be transmitted to the anthropoid apes. The specific infectious agent is Treponema pallidum, which may be demonstrated in the blood and in the lesions.

Acquired syphilis is usually, but not always, transmitted by sexual intercourse, or perversions thereof. But the disorder is not always a lues venera, since it may be transmitted by way of the lips in kissing, and by way of even slight abrasions or wounds of the skin or mucous membranes, such as of the fingers and hands of physicians, surgeons, obstetricians, and nurses, of the breast of wet nurses (syphilitic child), etc.; and it may also be transmitted indirectly by means of divers fomites, such as infected drinking and eating utensils, pipes, dental instruments, clothing, tattooing instruments, etc., and formerly by vaccine virus when human lymph was employed. The blood and the secretions from the primary, secondary, and tertiary lesions containing the specific infectious agent may transmit the disease if they come in contact with an abraded skin or mucous membrane. The normal

physiological secretions, such as the milk, tears, sweat, and urine, are ordinarily non-infectious, but they readily become infectious through contamination with the secretions of a syphilitic sore; the saliva and the semen are usually infected (through contamination).

Congenital syphilis may be transmitted in one of several ways: (1) By the father (sperm transmission), the mother being healthy. This is very common, the semen carrying the infectious agent, and infecting as it fertilizes the ovum. It is more likely to occur the more recent the infection in the father; it is unlikely after the lapse of three or four years, if the man has been efficiently treated; but it may occur even after years of apparently complete recovery and entire absence of symptoms. On the other hand, a man with florid syphilis may beget a healthy child. (2) By the mother (germ transmission), the father being healthy. The circumstances influencing the likelihood of infection are similar to those that obtain in the father; but a syphilitic woman almost always transmits the disease to her offspring, and this form of infection is much more fatal to the fœtus than is the paternal, since not only is the ovum diseased, but also the source of nourishment of the fœtus. A mother of a syphilitic child, though herself not obviously syphilitic, cannot become infected (having acquired an artificial immunity), as is well expressed in Colles' law: A child born of a syphilitic mother who is without obvious venereal symptoms, and which, without being exposed to any infection subsequent to its birth, shows this disease when a few weeks old, will infect the most healthy nurse, whether she suckle it, or merely handle and dress it; and yet this child is never known to infect its own mother, even though she suckle it while it has venereal ulcers of the lips and tongue. (3) By both parents being syphilitic. This is very common, one parent having infected the other—in which event the likelihood of infection in the fœtus is much enhanced. (4) By intra-uterine or placental transmission of the infection. A woman infected during pregnancy may, by way of placental transmission, infect her fœtus; this is less likely to occur the older the fœtus at the time of infection, and it is uncommon after the seventh month; but during birth the fœtus may acquire the infection from vaginal lesions.

A person having acquired syphilis is immune to a second infection, though occasional exceptions to this rule seem to be observed; and children who have inherited syphilis are immune to the acquired infection (Profeta's law).

Pathology.—The primary lesion, or chancre, consists of serous and cellular infiltration (lymphoid, plasma, epithelioid, and occasionally giant cells), especially in the deeper layers of the skin or mucous membrane about the capillaries and small veins, together with proliferation of the vascular endothelium, thromboses of the small vessels, and hyperplastic and degenerative changes in the small nerves. Together these constitute a firm, hard, reddish papule—the hard chancre. Soon, on account of deficient blood supply (due to the thromboses), liquefac-

tion necrosis sets in, and this, involving the overlying and perhaps proliferated epithelium, leads to vesiculation and usually ulceration—the ulcer being circumscribed, "scooped out," and indurated at the base and about the edges. Ultimately the serous and cellular infiltration subsides and is removed; fibroblasts form and cicatrization ensues. The lymph nodes adjacent to the primary lesion, and later the other lymph nodes throughout the body, become enlarged and indurated (buboes), in consequence of serous infiltration and cellular proliferation, and those adjacent to the chancre sometimes undergo suppuration in consequence of secondary pyogenic infection. Later all the lymph nodes exhibit more or less well-marked sclerosis. The cutaneous and the mucous membrane lesions consist of proliferation and infiltration of the upper layers of the skin and the mucosa, in some cases associated with cellular infiltration of the rete Malpighii and proliferation of the underlying bloodvessels. The characteristic mucous patch (mouth, genitals, anus) and the similar condyloma (of the soft and usually moist skin of the perineum, groins, axillæ, between the toes, etc.) exhibit lesions quite like those of the chancre—cellular infiltration of the deeper layers of the epithelium and elongation of the papillæ, proceeding to necrosis and exfoliation of the superficial layers. Later in the course of the disease diffuse indurative changes occur in the bloodvessels (arteriosclerosis, atheroma); these lead to or are associated with indurative changes in the organs (liver, spleen, kidneys, heart, brain, spinal cord, meninges, etc.). At the same time characteristic formations, so-called gummas (syphilomas, specific granulation tumors) appear, especially in the viscera, bones, periosteum, etc. These vary in size from so-called miliary gummas to formations ten to fifteen or more centimeters in diameter. They are firm, nodular, somewhat elastic, and on section gelatinous (or mucoid), and surrounded and pervaded by bands of dense, fibrous, connective tissue that also sends prolongations into the adjacent tissue. Occasionally considerable softening occurs, and if the gumma be situated in the skin or the mucous membrane small ulcers may form; these usually, sooner or later, cicatrize. Microscopically, the gumma consists of epithelioid and round cells and some giant cells, together with proliferation of the vascular endothelium, the formation of new bloodvessels, and a general tendency to sclerosis—although softening (mucoid and fatty degeneration) is quite common in the larger gummas. The so-called parasyphilitic lesions (syphilitic in origin but not in nature) exhibit no specific features.

Symptoms of Acquired Syphilis.—The period of incubation varies from two to five weeks, being about three weeks in the majority of cases. Usually there are no prodromal symptoms, the first indication of the infection being the so-called hard, or Hunterian, chancre. This develops at the seat of inoculation—usually in men on the prepuce or glans penis, though sometimes in the urethra (where it may remain unobserved), and in women on the vulva, the vagina, or the cervix of the uterus. The chancre, however, may be extragenital—on the

anus, rectum, lips, tonsils, breast, finger, etc. It consists of a firm, hard, painless, reddish papule, which soon exhibits slight central necrosis and ulceration, and may discharge a small amount of secretion, which sometimes dries to form a scab. By the third week the chancre has become characteristically hard, indurated, and cartilaginous; but this is more apparent on cutaneous than on mucous surfaces. At first it evinces no tendency to heal; but in most cases cicatrization is complete by the end of the fifth or sixth week, although it may be much delayed; the scar, as a rule, remains indurated for a long time. During the second week the lymph nodes adjacent to the primary lesion (usually, therefore, the inguinal nodes) become enlarged, hard, and indurated (syphilitic bubo); they are painless unless secondary pyogenic infections leading to suppuration occur. By the end of the third or fourth week the lymph nodes in general (epitrochlear, axillary, cervical, auricular, submaxillary, etc.), are enlarged and indurated.

The period prior to the development of the cutaneous and other so-called constitutional symptoms is usually spoken of as the primary stage, and lasts from six to twelve weeks. With the onset of the cutaneous manifestations the secondary stage is said to begin; this lasts from two months to a year or more, and is sometimes followed after a variable and uncertain period (often of apparently good health) by the so-called tertiary stage (gummas, certain cutaneous eruptions, amyloid degeneration, etc.); but no sharp line of demarcation between the secondary and the tertiary stage can be drawn: indeed, certain of the so-called tertiary manifestations sometimes develop unusually early, even before the chancre has healed. Nor is it correct to assume constitutional involvement only with the onset of the cutaneous and other manifestations of the so-called secondary stage—constitutional involvement is effected even before the maturation of the chancre (treponemæ may be demonstrated in the blood).

General Symptoms.—Slight fever is not uncommon with the onset of glandular enlargement, and it is almost always present when the glandular enlargement has become universal. In other cases no noteworthy fever may be apparent until three or four weeks before, or concurrently with, the development of the cutaneous lesions. This "fever of invasion" (as well as the attacks of fever that may occur at any time during the secondary or tertiary stages) may be moderate (101° F.) or sometimes quite high (104° F. or more); and it may be continuous (when it is likely to be mild), or remittent (usually), or intermittent (when it may be accompanied by chilly sensations or chills). This fever may be mistaken for typhoid fever, malaria, tuberculosis, sepsis, rheumatism, etc. In addition to the fever, evidences of general infection are seen in malaise, languor, prostration, general neuromuscular pains, bone pains (especially tibia, clavicle, and skull), joint pains, headache, iritis (exceedingly painful), coated tongue, poor appetite, slight enlargement of the spleen, and anemia (oligocythemia, disproportionate ologochromemia, and lymphocytosis). Late in the

disease aggravation of the anemia, cachexia, and amyloid degeneration may be observed.

Cutaneous Lesions.—The cutaneous lesions (syphilides), usually looked upon as marking the beginning of the secondary stage, are characterized by their polymorphism, superficial character, general symmetry, copper-colored hue, and absence of itching. Erythematous, papular, pustular, squamous, and tubercular syphilides may be distinguished. The commonest and the earliest is the roseola, which develops about eight to ten weeks after infection and consists of light reddish or rose-colored (later slightly coppery), roundish or ovoid macules, varying from 5 to 10 mm. in diameter, and irregularly distributed on the thorax and abdomen, sometimes on the thighs and arms (especially the flexor aspects), and rarely on the face. It persists for a week or two or longer; and may recur frequently. The syphilitic roseola may be accompanied, and is often followed, by papular and pustular syphilides, such as the so-called lenticulopapular, acneiform, varioliform, echthymaform, etc. These are usually distributed in groups over the trunk, flexor surfaces of the extremities, and the face, especially the forehead (corona veneris). A papulosquamous, so-called psoriasiform syphilide, is not uncommon; it may be circumscribed or widespread, and is rather common on the palms and soles. It may be distinguished from psoriasis by its coppery hue, absence of silvery scales, and absence of bleeding upon removing the scales. The soft and usually moist skin of the perineum, anus, vulva, groins, axillæ, angles of the mouth, between the toes, etc., is often the seat of characteristic condylomas—flat, well-defined, wart-like growths. Concurrently with the roseola the hair usually becomes thinned, lustreless, and falls out (alopecia). It usually returns upon appropriate treatment, except in the event of ulceration and cicatrization, when the alopecia is permanent. Onychia and paronychia are rather common (more common in congenital syphilis), and often lead to brittleness and shedding of the nails. Late in the course of the disease different gummatous lesions of the skin occur, of which the lupiform syphilide and rupia are the most important—since they give rise to considerable ulceration and destruction of tissue. Small roundish or irregular whitish spots (leukoderma) also are sometimes observed, especially in women and about the neck; they are usually 2 to 5 mm. in diameter, but they may coalesce to form larger spots or bands (collier de Venus), or they may spread over a greater part of the body.

Mucous Membrane Lesions.—The mucous membranes may be involved early or late, and the lesions may be erythematous, papular, ulcerative, or gummatous. Concurrently with the cutaneous rash, sometimes earlier, complaint is often made of sore throat, and on inspection the pharyngeal, faucial, and tonsillar mucous membrane is found swollen, red, and hyperemic; small papules soon form, and the superficial epithelium becoming opaque and cloudy, and later ulcerated, gives rise to the characteristic mucous patch—a moist, whitish, flat,

superficial, somewhat raised, and well-defined area. Such mucous patches may be found on any mucous membrane or mucocutaneous junction of the body, although they are most common in the fauces, pharynx, uvula, cheek, tongue, lips, nose, larynx, anus, vagina, etc. Sometimes these patches coalesce or spread to form more or less extensive serpiginous ulcers, especially on the tonsil, uvula, soft palate, and pharynx. Hypertrophy of the papillæ of the tongue is common early, and gives rise to papules which sometimes become necrotic and form small ulcers, especially near carious teeth; or ichthyosis may supervene; or the tongue may become atrophic, and the base, firm, hard, and smooth (Virchow's smooth atrophy of the base of the tongue)—which is of considerable diagnostic significance in persons over fifty years of age. Gummas are quite common in the mucous membranes and submucous tissues. In the tongue they not infrequently give rise to extensive ulceration and loss of tissue, and they have often been mistaken for carcinoma (and the tongue in consequence has been excised).

Circulatory System.—The chief lesions consist of arteriosclerosis and its consequences, notably fibroid degeneration of the myocardium, aneurysm, and aortic valve disease, all of which are discussed under their respective headings. A special type of arteriosclerosis due to syphilis may be distinguished, and in the lesions, especially in the aorta, Treponema pallidum may be found. Occasionally a gumma forms in the myocardium, and if situated in the pathways of the bundle of His may give rise to heart block. Syphilitic endocarditis is very rare (aside from arteriosclerosis); syphilitic pericarditis is perhaps more common.

Respiratory System.—The mucous membrane lesions in the nose (syphilitic rhinitis) have already been mentioned. In addition, gummas may occur, comparatively early. Usually they involve the bone before the mucous membrane; they give rise to considerable necrosis and destruction, to the occurrence of perforation of the hard and the soft palate and the bones and cartilages of the nose, and the consequent development of the so-called saddle nose and extreme fetor (ozœna). In the larynx, in addition to the common laryngitis, gummas may occur, and give rise to ulceration of the cartilages, with subsequent cicatrization and stricture. Involving first the epiglottis, they may cause a severe, even fatal, œdema of the glottis. Similar lesions may occur in the trachea or one of the main bronchi (dyspnœa, inspiratory stridor, lessened expansion and feebler breath sounds on the affected side); bronchial dilatation may occur below the site of the lesion. Syphilis of the lung is very rare: it may occur as small multiple gummas, about which there may be areas of bronchopneumonia; or it may lead to an interstitial fibrosis, developing, as a rule, from the root of the lung (perhaps very rarely from the pleura), and being especially conspicuous about the bronchi and the bloodvessels; the bronchi may become dilated. The diagnosis can rarely be made, although it may be suspected upon the development in a known syphilitic subject of the physical signs of chronic interstitial pneumonitis with bronchiectasis of the

lower lobe, and the absence of tubercle bacilli from the sputum; but one must remember that tuberculosis of the lungs may occur in a syphilitic subject, and tubercle bacilli may not be found for some time. A positive Wassermann reaction may be of diagnostic assistance.

Digestive System.—Rarely a gumma may develop in the œsophagus and ultimately lead to stricture. An occasional syphilitic ulcer has been observed in the stomach, but otherwise syphilis of the gastro-intestinal tract is rare, except in the rectum. Gummas are quite common in the rectum, especially in women. They develop usually just above the internal sphincter, and, gradually cicatrizing, produce a stricture. The symptoms are those of gradually increasing difficulty in evacuating the bowels and narrowing of the fecal masses. The stricture may be recognized by palpation and protoscopic examination, and may be distinguished from carcinoma by its being firm and fibrous rather than a more or less exuberant, centrally ulcerated mass, and by the absence of the carcinomatous cachexia.

Syphilis of the liver is very common. Jaundice is not uncommon at the time of development of the cutaneous rash and enlargement of the lymph nodes; it is likely due to radicular cholangitis. Sometimes a type of acute yellow atrophy supervenes. In the tertiary stage, the lesions may consist of: (1) Gummas, which may be single or multiple; usually they give rise to a large protruding tumor in or near the epigastrium, together with local pain and distress. The diagnosis is facilitated by the detection of evidences of syphilis elsewhere. Carcinoma may be eliminated by the less rapid growth or stationary condition of the enlargement (growth is usually rapid and progressive in carcinoma), and by the absence of the general, cachectic, manifestations of carcinoma. In some cases, however, the therapeutic test is necessary to establish the diagnosis. The presence of fever may suggest liver abscess, but the other evidences of suppuration are usually absent. Following absorption of the gumma the liver becomes more or less distorted from scar formation. (2) Chronic interstitial hepatitis. These cases exhibit the common manifestations of cirrhosis—enlargement of the liver with the signs of portal obstruction and ascites; there is likely to be also slight jaundice and some fever, at least from time to time; later the liver may decrease in size. Perihepatitis is usually associated, and may give rise to palpable and audible frictions. The lesions may also implicate the peritoneum more extensively. (3) Amyloid disease presents the symptoms and signs mentioned under amyloidosis.

Genito-urinary System.—In a few cases an acute syphilitic nephritis occurs during the second stage of the disease; death may ensue soon, but, as a rule, recovery follows after a number of months, although albuminuria may persist for a long time. A chronic parenchymatous nephritis may be associated with the renal and other lesions of amyloidosis. Chronic diffuse indurative (or interstitial) nephritis is a common sequence of syphilis and the arteriosclerotic changes that it induces. Miliary or larger gummas may be found in the kidney, espe-

cially in association with syphilis of the liver. The testicle may become the seat of gummas or of progressive fibrosis (interstitial orchitis). Abortion and repeated abortion is common in syphilitic women.

Nervous System.—The lesions of the brain may consist of: (1) Syphilitic arteriosclerosis, which may lead to aneurysm and rupture of a vessel (apoplexy), or to thrombosis and cerebral softening. (2) Gummas, which may be single or multiple, and vary in size from that of a pea to that of an egg. They may occur in any part of the brain, but usually develop in the dura mater or the pia-arachnoid at the base of the cerebral hemispheres, near the pituitary body, or on the convexity of the frontal convolutions. When the meninges are involved, a subacute or chronic meningitis with much thickening results, and the lesions are likely to extend along the nerve sheaths (especially the oculomotor, the facial, etc.). The symptoms of the larger gummas are those of tumor of the brain—especially headache, vomiting, convulsions, optic neuritis, etc., together with various localizing symptoms depending upon the situation of the gumma. (3) Meningitis—which may occur without definite macroscopic gummatous formations, although microscopic gummas are almost always present. This is common at the base of the brain, where it usually implicates one or more of the cranial nerves, especially the oculomotor, the trochlear, the abducens, and the trigeminal; but it may involve also the cortex. The chief symptoms consist of initial headache, which may be local or general, and constant or intermittent, but is usually worse at night; vertigo, somnolence, failure of mentality, mental depression, sometimes irritability, and localizing symptoms depending upon the cranial nerves involved. In some cases there is eventually coma or convulsions; rarely these may be the major initial manifestations. The symptoms due to cortical involvement are those of meningo-encephalitis, which gradually progress to well-defined paretic dementia.

Spinal cord lesions are less common than cerebral; they may consist of arterial disease (with softening of the cord); or gumma (tumor formation) of the meninges or the cord; or meningitis (which usually is irregularly distributed, although it involves all the meninges and the spinal cord and the spinal nerve roots); or of myelitis and secondary sclerosis. The so-called parasyphilitic (metasyphilitic) diseases involve the nervous system, and consist of tabes dorsalis, paretic dementia, etc. The diagnosis of cerebrospinal syphilis, in some cases readily made, may be difficult; the symptoms are likely to be variable, multiform, and asymmetrical, to exhibit combinations of spinal, cerebral, and meningeal symptoms not readily explainable by a single or well-defined lesion. In some cases resort must be had to antisyphilitic treatment to settle the diagnosis.

The Bones, Joints, Bursæ, and Muscles.—Syphilitic periostitis is common, and gives rise to local pain, often worse at night, tenderness, and swelling. Gummas may develop in the periosteum as well as in the deeper structures, reaching even to the medulla and causing

considerable destruction. Occasionally acute or chronic synovitis is observed; or a syphilitic osteo-arthritis may occur. The muscles may be the seat of an early myositis, or of a later gummatous formation. Absorption of the gumma may lead to more or less loss of substance and fibrosis. The syphilitic bursopathy of Verneuil, occurring especially about the larger joints (knee, elbow, etc.), is a rare manifestation.

Symptoms of Congenital Syphilis.—Aside from the primary sore, congenital syphilis presents all the manifestations of the acquired disease. The child at birth may present obvious evidences of the disease, or it may appear healthy, the symptoms not developing until about the second month; in other cases, the symptoms first become obtrusive about puberty (delayed congenital syphilis). The infant obviously syphilitic at birth is poorly nourished, wizened, old-looking; it suffers from coryza that interferes with respiration (whence "snuffles") and with suckling; the lips and the angles of the mouth are fissured and ulcerated, and blisters are present on the hands, wrists, feet, and ankles (pemphigus neonatorum). The liver, spleen, and pancreas are usually enlarged; the lungs are heavy, firm, grayish white, and almost, if not quite, airless (the so-called white pneumonia of Virchow); and the ends of the long bones commonly reveal an osteochondritis that may lead to separation of the epiphyses. Hemorrhages, subcutaneous, submucous, umbilical, occur in some cases. These infants, as a rule, soon die; fœtuses born dead reveal the same changes.

In other cases the infant is apparently healthy at birth. Usually early in the second month the syphilitic rhinitis ("snuffles") develops; the discharge may be serous, or purulent, or sanguinolent, and is highly infectious. Necrosis and ulceration of the nasal bones and cartilages may ensue, and lead to falling in of the nose—the so-called saddle-nose; or the lesions may spread to the pharynx, the Eustachian tube, and the middle ear, and give rise to otorrhœa and deafness. Cutaneous lesions quite analogous to those of the acquired disease develop; usually they begin about the genitals and buttocks, but tend to spread over the body. The most common is a yellowish or reddish-brown erythema, but the lesions may be also papular, eczematous, or pustular. Fissures (ragades) are common about the lips, especially at the angles of the mouth. Mucous patches, condylomas, alopecia, onychia, dactylitis, iritis, etc., also occur; the liver and spleen are usually enlarged; the bones about the fontanelle and elsewhere may be thickened, and the epiphyses may be enlarged. Many of these children also die; but in favorable cases and under appropriate treatment, the symptoms subside toward the end of the first year, and the child may appear perfectly healthy until about puberty. From puberty to about the twentieth year many so-called late manifestations of congenital syphilis occur. As a rule, growth is retarded, and the child appears wizened and prematurely old; the skull may be large or asymmetrical, the nose depressed, the lips prominent, and scars of healed fissures may radiate from the corners of the mouth—appearances which of them-

selves suggest the disease. The teeth are likely to be deformed, and the two upper central incisors may be short and broad, peg-shaped (narrower below than above), and reveal a single notch at the free edge (Hutchison's teeth). Interstitial keratitis (ground glass opacity of the cornea), usually bilateral, and bilateral deafness, unassociated with pain or discharge, are quite common. The malformations of the teeth, interstitial keratitis, and deafness constitute Hutchison's triad of congenital syphilis. Bone lesions also are common—especially bilateral gummatous periostitis, affecting especially the tibiæ, but also other bones, and leading to permanent thickenings and deformities. Joint lesions are rare, although a synovitis, usually symmetrical and involving the knee by preference, may occur. Gummas in different parts of the body and amyloid disease are rare manifestations.

Diagnosis.—In some cases the initial lesion is so characteristic as to admit of no doubt of the diagnosis; in other cases in the past, certainty was not obtainable until the development of enlarged lymph nodes and the cutaneous rashes. But at present Treponema pallidum may be readily demonstrated in the serum of the chancre (by "dark-field" illumination or by staining), as well as in a section excised from the chancre, and in the circulating blood, so that it is no longer necessary to await the tedious appearance of the so-called secondaries to make a positive diagnosis and institute treatment. In older cases in which the diagnosis is uncertain, aid may be obtained by a history of a primary lesion or of exposure to infection, but both may be denied; there may be, however, a history of sore throat, cutaneous rashes, alopecia or iritis, etc., and in women of repeated abortions; search may reveal the scar of a primary lesion or of other syphilitic lesions elsewhere, or copper-colored cicatrices on the legs (not always syphilitic), or periosteal thickenings, or enlarged lymph nodes, etc. Congenital syphilis in early infancy is readily recognized from the general appearance of the child, the snuffles, the cutaneous eruptions, the ragades at the angles of the mouth, the condylomas, enlarged liver and spleen, etc. Snuffles and a rash occurring within the first three months of life are almost certainly syphilitic. There may be also a history of syphilis in one or both parents. The disease manifesting itself about puberty may be recognized by the retarded growth and general appearance of the child, Hutchison's teeth, usually bilateral keratitis, bilateral deafness, symmetrical nodosities on the bones, etc. In any case of supposed congenital or acquired syphilis, or of parasyphilitic disease, such as tabes dorsalis, paretic dementia, arteriosclerosis, aortic valvular disease of the heart, etc., resort may be had to the Wassermann reaction, which seems to give positive results in 80 to 90 per cent. of the cases. Finally, in doubtful cases, resort may be had to the therapeutic test, which, however, is by no means infallible.

Prognosis.—In acquired syphilis, the outlook is good if treatment is instituted early and carried out efficiently and for a sufficiently long time—which is rarely the case. Some cases are unusually virulent,

and more or less rebellious to treatment; and in other cases a fatal acute yellow atrophy of the liver may supervene. Death is frequently due to tertiary, gummatous lesions, and to related disorders due to arteriosclerosis, etc. The prognosis in the parasyphilitic diseases, tabes dorsalis and paretic dementia, is almost hopeless as to cure. Congenital syphilis may lead to death of the fœtus or to death soon after birth, hemorrhages, jaundice, and pemphigus especially being bad omens. The ocular and aural manifestations developing about puberty may be more or less permanent. There is considerable doubt whether syphilis can be transmitted to the third generation.

Treatment.—The only efficient method of prophylaxis is to avoid the sources of infection. A syphilitic subject should be told of the danger of transmitting the disease by kissing, for instance, as well as by contaminating any article that he may use, such as table utensils, pipes, public drinking vessels, personal and bed linen, towels, etc. Physicians, nurses, midwives, barbers, chiropodists, etc., should carefully sterilize all instruments, towels, etc., that have been used on known or supposedly syphilitic persons. A syphilitic infant may be nursed by its mother, but should not be given to a healthy wet nurse, who certainly would acquire the infection from the infant. From a purely medical point of view, altogether aside from the question of morals involved in exposing an innocent and unsuspecting woman and possible children to the likelihood of infection, a syphilitic subject must not marry until at least four years have elapsed since infection; the first two years must have been devoted to persistent and efficient treatment, and during the second two years the subject must have been entirely free from symptoms; otherwise the physician must prohibit the marriage, and such quasi approval as he may give should be always reserved, since even after apparently efficient treatment infection may be transmitted, and late results, such as gummas, tabes dorsalis, and paretic dementia, are likely to ensue.

Opinions differ as to the propriety of excising the initial lesion with a view to prevent constitutional symptoms. Although it is undoubtedly true that general infection has occurred by the time the chancre has matured, it is perhaps possible to minimize the infection by early removing the main hatching ground; this, however, can scarcely be proved, although it has been successful in the ape. Treponema pallidum, however, should first be demonstrated in the chancrous discharge. If not removed, the chancre should be washed with an antiseptic solution, such as mercuric bichloride 1 to 2000, and dusted with calomel; or a calomel ointment (10 per cent.), or mercurial plaster, may be applied.

Internal medication should be commenced as soon as the diagnosis is certain, and not sooner. If Treponema pallidum has been early identified, treatment may then be instituted; otherwise, one should await the general lymph-node enlargement, the cutaneous rash, and other so-called secondaries. Mercury is the sovereign remedy, and may be given by the mouth, by inunction, by intramuscular injection, and

by fumigation. The preferable preparations for administration by the mouth are: Mercury with chalk (containing 38 per cent. of mercury), which may be given in doses of 1 grain (0.06 gram), with or without an equal amount of Dover's powder to prevent diarrhœa in those susceptible; the protoiodide ($\frac{1}{4}$ grain, 0.015 gram), the tannate ($\frac{1}{4}$ grain, 0.015 gram), and the biniodide ($\frac{1}{16}$ grain, 0.004 gram), three times a day. The dose may be increased to the point of toleration (abdominal pain, diarrhœa, tenderness of the gums), whereupon the dosage should be reduced one-half, and continued thereat for one year, provided the general health of the patient is maintained. Should the general health fail, the mercury should be stopped, and iron and tonics substituted. Should relapses occur while taking the mercury, the dose should be increased, or another method of administration resorted to. During the second year, in the absence of symptoms, there should be alternating periods of treatment and of cessation of treatment of a month or six weeks each; if symptoms are present, treatment should be continuous, as in the first year, and the alternate periods of treatment and cessation thereof should be resorted to in the third year.

In cases in which a rapid influence is desired, inunctions may be resorted to. After preliminary cleansing, 1 dram (4 grams) of the official mercurial ointment (50 per cent. of mercury), or an equivalent amount of the oleate (10 per cent. of mercury), should be rubbed into the body daily (for 20 to 30 minutes) on six consecutive days; on the seventh day the patient should have a warm, cleansing bath. The skin of the parts selected for the inunction should be thin and as free from hair as possible: the inner aspect of the thighs, the sides of the chest and abdomen, and the arms are six serviceable regions that may be used in rotation. When the inunction is done by some one other than the patient, the rubber should be protected by gloves or he should use a glass rod or spatula. The treatment should be continued for four or five weeks, and then discontinued; but at least two courses of such treatment should be given yearly for two years, or longer should symptoms recur. Intramuscular injections are exceedingly valuable in intractable cases, in cerebrospinal syphilis, and in persons who dislike the inconvenience and publicity of pills and inunctions. One may use: (1) The insoluble salts of mercury, such as calomel, 1 grain (0.06 gram), in 15 minims (1 c.c.) of sterile olive oil; gray oil, 1 to 2 minims (0.06 to 0.12 c.c.) diluted with olive oil; or mercury salicylate, 10 minims (0.60 c.c.) of a 10 per cent. suspension in paraffin oil; or (2) the soluble salts of mercury, of which the bichloride is the best, and may be used in doses of $\frac{1}{8}$ grain (0.008 gram) in 15 minims (1 c.c.) of sterile physiological saline solution. The injections should be made deep into the buttock, in the centre of a line running from the anterior superior spine of the ileum to the upper end of the intergluteal fold. The insoluble salts should be injected once a week for twelve to fifteen weeks, and the soluble salts, at first daily, then every other day, and then twice a week until about forty injections have been made. The

injections may then be discontinued for a month or two, but, as in the other methods, treatment must be resorted to for shorter periods for at least two years, and again if any symptoms recur.

In the late or tertiary stage of the disease (visceral and bone lesions) the iodides should be given, either alone or alternating with or in combination with mercury (so-called mixed treatment). The iodide may be given in gradually ascending doses, in some alkaline water or milk; although excellent results sometimes attend the use of small doses, 15 grains (1 gram) daily, it is wise, as a rule, to give large doses to the point of toleration (150 grains, 10 grams, or more a day). One may give the iodide and mercury together, as in the following prescription (the arsenic has been added for its tonic effect and to prevent acne, coryza, and other manifestations of iodism, but it may be omitted):

℞—Mercuric bichloride	1 grain	0	6
Potassium iodide	3 drams	12	0
Solution of potassium arsenite	30 minims	2	0
Compound syrup of sarsaparilla	4 ounces	120	0
Water sufficient to make	8 ounces	250	0—M.

Sig.—One tablespoonful (15 c.c.) in water three times a day.

The iodides should be continued for two or three months, then intermitted for a month, and then resumed, alternating thus month by month for a year. Mercury is sometimes administered also by fumigation (sublimation of calomel), and, rarely, by intravenous injection. At all times when mercury is being taken the greatest care must be exercised to keep the mouth clean, by frequent use of the toothbrush, and of solutions of potassium chlorate or mild antiseptics. Alcohol and tobacco should be interdicted. Mucous patches may be treated with solid silver nitrate, acid nitrate of mercury, etc.; condylomas about the genitalia by cleansing with mercuric bichloride, 1 to 2000, and dusting with calomel; scaly lesions on the hands, feet, etc., by a 10 per cent. ointment of ammoniated mercury; and ulcerating cutaneous gummatous lesions by cleansing and the application of mercurial plaster. The syphilitic cachexia should be treated with bitter tonics, iron, quinine, strychnine, syrup of iron iodide, cod-liver oil, arsenic, the lactophosphates, and hypophosphites.

Syphilitic infants are best treated with mercurial ointment (diluted with an equal part of vaseline) or the oleate of mercury on the binder, or by administering mercury and chalk, ½ grain (0.03 gram), three times a day. The later lesions occurring about puberty may be treated with mercury and the iodides, the following formula being serviceable:

℞—Mercuric bichloride	½ grain	0	03
Potassium iodide	1½ drams	6	0
Tincture of iron chloride	2 drams	8	0
Syrup of lemon	2 ounces	60	0
Water sufficient to make	4 ounces	120	0—M.

Sig.—One teaspoonful (5 c.c.) in water three times a day.

YAWS.

(*Frambœsia.*)

Yaws is a chronic, infectious, and highly contagious disease, caused by Treponema pertenuis (Castellani), and characterized by a papular rash which usually develops into a fungating, granulomatous, encrusted eruption.

Etiology.—Yaws is widely distributed throughout the tropics, especially Africa, Asia, Central and South America, the Pacific Islands, and the West Indies. During the early part of the nineteenth century it was exceedingly common among the slaves of our southern countries, but has now practically disappeared from the land. The disease attacks persons of all ages, but children are most susceptible. Indeed, in some parts of the West Indies and in Fiji nearly every child is attacked, and those who might otherwise escape are often voluntarily exposed to infection by their parents under the impression that the disease is a necessary part of a child's training. The black and the yellow races are more susceptible than the white. One attack seems to confer partial immunity. The disease is not hereditary or congenital, as is syphilis, for which it is sometimes mistaken.

The exciting cause of the disease is Treponema pertenuis. The disease is highly contagious. The contagium may be transmitted directly, that is, from the sick directly to the well; by means of fomites, such as articles of domestic use, dishes, etc.; and probably also by insects; but an open wound or abrasion seems requisite for infection. Mere residence in an infected locality (a house, for instance) seems to have sufficed for infection, but many other factors must be considered in such cases. The infection is inoculable into lower animals, such as the monkey.

Symptoms.—The period of incubation varies from about two weeks (in inoculation cases) to as much as six months (in cases otherwise acquired). Prodromal symptoms are usually absent, though there may be for several days malaise, headache, fever, and vague neuromuscular pains. The disease begins, certainly in the inoculation cases, and usually also in other cases, with the development of a papule at the site of inoculation—usually on the feet, legs, or face. At the end of a week this is converted into an ulcer, which at the end of another week heals and leaves a superficial cicatrix. This is called the primary eruption or sore. The secondary eruption, which may occur with the primary eruption, or be delayed for two weeks, consists of pin-sized papules which gradually increase in size until they reach that of a pea or a small nut, or, by confluence, that of a small apple. They may be distributed more or less widely over the body, but are found most commonly at or about the mucocutaneous junctions and on the limbs, neck, and trunk; the hairy scalp and the axilla are rarely involved. They consist largely of granulation tissue and inflammatory overgrowth of the papillary layer of the cutis and the Malpighian layer of the epidermis. They much resemble

syphilitic condylomas, and itch considerably, but are otherwise painless. Their epidermic covering becomes thinner, and is ruptured or shed, permitting the escape of a whitish-yellow, seropurulent fluid which dries and forms crusts, which are usually quite tough, often very hard, and adherent. When removed they reveal irregular, reddish granulation tissue, which has been likened to a raspberry, whence the name frambœsia (*frambœsia*, raspberry). The yaw, as a rule, reaches its maximum development in about two weeks, remains stationary for about two weeks, and then begins to shrink. The crust dries up, the papillomatous-looking granulation tissue is gradually converted into a cicatrix, which is pale in the negro, pigmented in the white race, and surrounded by an area of increased pigmentation.

In some cases the primary eruption is followed by one or more relapses, associated with fever and severe neuromuscular pains in the back. In other cases the yaw itself ulcerates, but may heal after a time with considerable cicatricial contraction.

Diagnosis.—The disease is commonly unmistakable, but care must be exercised to exclude syphilis, verruga, leprosy, tuberculosis, and epithelioma. Treponema pertenuis may be demonstrated in the lesions, especially the papillomatous skin lesions.

Prognosis.—The duration of yaws is from a few weeks to several years. In most cases recovery ensues. Young children, however, and older persons the subject of divers constitutional diseases, may succumb.

Treatment.—Attention to general hygienic measures is imperative—isolation, fresh air, good food, suitable clothing (to obviate cold and chill), and daily warm baths. The ulcerated areas should be treated antiseptically and with stimulants, such as copper sulphate, silver nitrate, chromic acid, etc. Potassium iodide internally is believed to be of much value in effecting the disappearance of the local lesions. Mercury is sometimes used, but often avoided, as tending to do harm. The bitter tonics and iron, arsenic, and quinine are useful as general measures.

TRYPANOSOMIASIS.

(*Sleeping Sickness; African Lethargy; Tsetse Fly Disease.*)

Trypanosomiasis is a chronic, specific, infectious disease caused by Trypanosoma gambiense, and characterized by fever, weakness, lassitude, emaciation, and eventually by protracted lethargy—sleeping sickness.

Etiology.—Sleeping sickness is known to have existed for the last one hundred years on the west coast of Africa, and recently has been reported inland. It affects the white as well as the colored race, and it is common at all seasons. The specific exciting cause is Trypanosoma gambiense, which is transmitted by a tsetse fly, Glossina palpalis, which Koch believes becomes infected by feeding on infected crocodiles, and which probably serves as an alternate host; that is, the parasite probably undergoes some phase of development in the fly.

Symptoms.—The period of incubation is probably from two to three weeks. In the early stage of the disease, which varies in duration from about three to as much as three years, the only symptom of marked constancy is enlargement of the lymph nodes, but there may be also irregular fever, rapid pulse, mental hebetude, slight anemia, and some loss of weight. This stage gradually merges into the second stage, that of sleeping sickness. The patient becomes definitely dull, listless, and apathetic; the pulse becomes rapid (120 or more per minute) and weak; the temperature becomes irregular, often rising as high as 101° or 102° in the evening; indefinite pains are complained of in the head and throughout the body; patches of erythema or of œdema may develop on the skin. There is gradual loss of strength in the arms and legs, associated with tremors, as well as with tremors of the tongue. The patient lies most of the time in a lethargic state and becomes emaciated. Finally, during the last two or three weeks of life the temperature falls below normal, and the patient ultimately dies in coma.

Diagnosis.—The diagnosis may be made tentatively from a consideration of the symptomatology and possible etiological factors, and positively on finding Trypanosoma gambiense in the enlarged lymph nodes (aspiration of some fluid therefrom with a hypodermic needle and syringe), circulating blood, or cerebrospinal fluid. Darling has described, as occurring upon the Isthmus of Panama, a fatal infection resembling kala-azar, that he has named Histoplasma capsulatum, and due, as he believes, to an organism resembling Leishmania donovani.

Prognosis.—The disease is invariably fatal.

Treatment.—Prime importance must be attached to avoiding the tsetse fly, and searching out and destroying its breeding places. Treatment is unavailing, arsenic being the only drug apparently capable of producing any good effects, and it only prolongs life. The ordinary preparations or atoxyl may be used. They have sometimes been combined with aniline preparations, such as trypanroth, with asserted good results. Hopes are entertained of the production of an efficient serum.

KALA-AZAR.

(*Tropical Splenomegaly; Tropical Cachexia; Dum-dum Fever; Piroplasmosis.*)

Kala-azar is a chronic, specific, infectious disease caused by Leishmania donovani, and characterized by intermittent or remittent fever, marked and progressive anemia and emaciation and enlargement of the spleen and the liver. A similar organism, described as Leishmania infantum (Pianese and Nicolle), has been found in certain types of infantile splenic anemia.

Etiology.—Kala-azar was formerly believed to be indigenous in the lowlands of India, but it is now known to occur in many regions of the Orient. How the disease is transmitted is not definitely known. Patton has studied the flagellated stage of Leishmania donovani in the bed-bug

(Cimex rotundatus), but whether this is a transmitting agent has not yet been determined.

Symptoms.—The period of incubation is not definitely known, but it may be less than ten days (Manson). The onset of the disease is usually abrupt—chills, fever (104° to 106°), vomiting, and rapid enlargement of the spleen and liver. The fever, though somewhat irregular in type, remains high for a few days to six weeks, and then subsides by lysis to normal or subnormal; recurrences are common, and a low grade continued fever may supervene. Profound debility, emaciation, and anemia (rarely less than 2,500,000 erythrocytes) and marked leukopenia (involving especially the polynuclear neutrophiles) ensue. The skin becomes peculiarly pigmented—of an earthy gray or drab color; and subcutaneous hemorrhages and dropsical effusions may develop in the skin and the serous sacs. The disease lasts for several months, as a rule, but may be prolonged to one or even two years.

Diagnosis.—The diagnosis is suggested by continued fever, enlargement of the spleen, leukopenia (relative lymphocytosis), and is confirmed by detecting Leishmani donovani in the blood (especially in the leukocytes) or in the tissues.

Prognosis.—Death occurs in about 96 per cent. of the cases—from exhaustion, or intercurrent infections, such as chronic diarrhœa, etc.

Treatment.—The treatment is purely hygienic, dietetic, and symptomatic. Removal from infected regions and isolation of the patient are advisable.

ORIENTAL SORE.

(Tropical Ulcer; Delhi Boil.)

Oriental sore is a chronic, specific, granuloma that occurs endemically in certain tropical and subtropical countries, is caused by Leishmania tropica (Helcosoma tropicum, Wright), and is characterized by a nodule, which breaks down and forms an indolent ulcer.

Etiology.—How the exciting cause of the disease is transmitted is not known, but the bite of an insect or infection of an already existing wound is usually postulated. Age, sex, race, and occupation are devoid of etiological significance. One attack seems to confer immunity.

Symptoms.—The disease begins with a small, red spot, resembling a mosquito bite, in the centre of which a small nodule or papule soon forms. The surface becomes covered with fine scales that form a crust, which, when removed or separated spontaneously, leaves a shallow, painless ulcer, that may itch considerably. About this similar ulcers develop, and by coalescence of many, a single ulcer, often an inch in diameter, results. In some cases there is only one, sometimes two ulcers, whereas in other cases there may be as many as twenty. After the lapse of from two to twelve months, healing begins. The resulting cicatrix may occasion considerable deformity, especially if on the face. Occasionally the nodule does not ulcerate, but merely desquamates at the

end of several months. The general health is little, if at all, disturbed. Enlargement of the lymph nodes, lymphangitis, phlebitis, erysipelas, abscesses, and gangrene are sometimes encountered as complications, and may result in death.

Diagnosis.—The diagnosis suggested by the symptoms, is confirmed by detecting the specific cause.

Prognosis.—The specific lesion itself rarely leads directly to death, but the prognosis as to cure is unfavorable.

Treatment.—Cauterization of the specific lesion is recommended by some observers. At all events it should be dressed antiseptically. Change of climate and tonics are said to be useful in some cases.

MALARIAL INFECTIONS.

(*Ague; Intermittent Fever; Remittent Fever; Pernicious Remittent Fever; Bilious Remittent Fever; Estivo-autumnal Fever; Paludism; Swamp Fever; Marsh Fever.*)

THE malarial infections comprise a group of specific infectious diseases, caused by Plasmodium malariæ (Hæmamœba malariæ, Laveran), and variously characterized by intermittent or remittent fever, enlargement of the spleen, the presence of the specific microörganism in the circulating blood, and sometimes by certain so-called pernicious manifestations or a chronic cachexia.

Etiology.—The malarial infections are widely disseminated throughout all parts of the world. Though especially prevalent in tropical and subtropical regions, they frequently prevail extensively in the temperate zones; but a noteworthy diminution in the prevalence of the infections is found to follow intelligent agricultural pursuits, efficient drainage, and other sanitary measures commonly introduced by the more enlightened people. On the contrary, the infections not infrequently break out among laborers engaged in constructing railroads, canals, fortifications, and other operations connected with upturning the soil. These and other etiological (telluric and atmospheric) factors, such as comparatively high atmospheric temperature and humidity, are of influence only in so far as they favor the growth and development of the Anopheles mosquito—the essential factor in disseminating the infections. The mosquito fructifies and flourishes best at a temperature of 75° F., or over —whence the malarial infections are most common in tropical and subtropical regions, and in temperate zones during the summer. Pools of stagnant water are essential for the breeding of mosquitoes—whence malarial infections are common in the neighborhood of earth-cutting operations accompanied by the formation of holes and surface irregularities wherein water collects and stagnates, and in the neighborhood of low marsh lands, swamps, and old river beds, where the water affords the necessary medium and the abundant vegetation additional protection for the developing larvæ. The malarial infections are less common at

high than at low altitudes, because the mosquitoes, as a rule, do not fly high; nor do they fly far (200 to 300 yards), although they may be carried long distances by winds; and the proverbial insalubriousness of the night air is attributable to the fact that the malaria-bearing mosquito (Anopheles) peregrinates only at night.

In this country the malarial infections at present are endemic along the Gulf of Mexico and the Mississippi River and in the States adjacent thereto, and they are rather common in the Middle and Southern States that border the Atlantic Ocean. They are much less common than formerly in the central, New England, and Northwestern States; and the Hudson, the Delaware, and the Schuylkill valleys, formerly much infected, are now comparatively free. The infections are uncommon in Canada. Many of the Central and South American States, especially those along the coasts, are markedly infected. The British Isles are practically uninfected. The valleys of lower Russia, the Balkan provinces, Italy, and India present many foci of infection, and along the coast of Africa the most malignant forms of the infections are endemic.

Age, sex, and race are devoid of etiological significance. The supposed relative immunity of the adult negro of South Africa has been found to be due to a relative immunity acquired during childhood, when the disease is very common, though often masked. Occupation is of significance only in that laborers on railroads, in swamps, etc., and soldiers, fishermen, farmers, etc., are more exposed to the likelihood of being bitten by infected mosquitoes.

The essential etiological factor, then, is infected mosquitoes. Thus far only mosquitoes of the genus Anophelinæ have been found to be malaria-bearing hosts, and of the one hundred and fifty or more species, forty have been proved by experiment to be active infectious agents. The most common and important are: Anopheles maculipennis (also called quadrimaculatus, and claviger—the most common), Anopheles punctipennis, Anopheles crucians, and Anopheles argyritarsis. Fortunately in the temperate zone the common mosquito is not Anopheles, but Culex—which does not transmit malaria (though it does transmit filarial disease). The two may be distinguished by the fact that the palpi of the adult Culex are very short, while those of Anopheles are almost if not quite as long as the proboscis; the wings of Anopheles are distinctly mottled (brownish), while those of Culex are not (aside from the veins); Culex while resting has its body nearly parallel with the surface (wall) and its posterior pair of legs turned up over its back, while Anopheles rests with its body almost at right angles (45 degrees or more) to the wall and its posterior pair of legs usually against the wall or hanging downward. Anopheles is a rural insect, breeding especially in shallow pools, irrigating ditches, and the still edges of running streams, often remote from human habitations; Culex is a city beast, breeding in tanks, cisterns, and stagnant pools of water near or in houses. The males of all mosquitoes have feathered antennæ (woolly heads), while the females have not.

The mosquito, like other insects, passes through the stages of ovum,

larva, pupa, and imago (adult insect). Depending upon the genus, and the environmental conditions, such as season, temperature, stillness of the water, food, etc., the cycle of development from ovum to imago takes from ten days to three or four weeks. The male mosquito thrives on the juices of plants, whereas the female mosquito feeds mostly upon the blood of vertebrates, a meal of which is said to be essential to the deposition of eggs. These, as a rule, are deposited on the surface of still water, and they vary in number with the species: Anopheles and Stegomyia deposit 40 to 50 ova, singly or in small groups, each ovum resting on its side; Culex deposits 300 to 400 ova, in large, coherent masses, the individual ova being on end. The ova are very resistant, and may mature even after several months of drying. In from twelve hours to three or four days, depending upon the temperature of the water and the air, the ova develop to larvæ—minute, whitish or brownish, worm-like bodies that hang below the surface of the water and exhibit an active wiggling motion, whereby (about once a minute) they come to the surface to breathe, through a respiratory tube that projects from the last abdominal segment. The larvæ may remain capable of maturation after several months of unfavorable environmental conditions, even freezing in ice. After existing in the larval stage for about one week (Culex and Stegomyia) to two weeks (Anopheles), during which time several moultings occur, the pupæ appear—distinguishable from larvæ chiefly by the greater size of the thoracic segment, but also by their disposition to float on the water (being lighter than water). At the end of from two days (Culex and Stegomyia) to five days (Anopheles), the imago or adult insect emerges from the pupal shell. The first deposition of eggs occurs about twenty days later, and recurs at intervals of several days—each female mosquito being said to give rise to at least twelve generations in a season. The Anopheles mosquito becomes infected with the malarial parasite by sucking the blood of a malarious subject.

Plasmodium Malariæ in Man.—Craig distinguished four distinct species: Plasmodium vivax (Grassi and Feletti), the tertian parasite; Plasmodium malariæ (Marchiafava and Celli), the quartan parasite; Plasmodium falciparum (Blanchard), the tertian estivo-autumnal parasite; and Plasmodium falciparum quotidianum (Craig), the quotidian estivo-autumnal parasite. Each species passes through two different cycles of development, the one in man, the other in the mosquito.

The Tertian Parasite.—The tertian parasite is seen in its earliest stage within the infected erythrocyte as a small (2μ in diameter), roundish, hyaline, unpigmented, and actively amœboid body—protruding and retracting pseudopodia. This increases in size and soon accumulates a number of fine, reddish-brown pigment granules which it has elaborated from the hemoglobin of the infected erythrocyte, and which, reflecting the amœboid movement of the parasite, appear actively motile. When almost or quite full grown the parasite is roundish, distinctly outlined, actively amœboid, and contains motile pigment granules which are usually distributed rather diffusely (sometimes in small clumps) through-

out the parasite; what are usually described as a nucleus and a nucleolus may be distinguished in stained specimens. The containing erythrocyte is much swollen and pale. At the end of thirty-six hours, as maturity is reached, scarcely any of the distended erythrocyte (which may be almost twice the normal size) is visible, the amœboid motion has lessened, and the pigment granules, much increased in number, tend to collect in the centre of the parasite. Soon radial striations appear and divide the parasite into from twelve to twenty-four (an average of sixteen) segments, which contain no pigment and are arranged usually in two rows, one toward the periphery and the other toward the centre, though they may be arranged irregularly. Toward the end of forty-eight hours the erythrocyte has been entirely destroyed, and segmentation of many of the parasites (sporocytes) occurs; the free segments, liberated in the blood stream, float about as unpigmented bodies, and soon re-infect other erythrocytes and again pass through this so-called asexual cycle of development. Some parasites, however, do not undergo segmentation, but persist as large parasites with actively motile pigment granules—the sexually differentiated microörganisms (gametocytes) designed for the sexual cycle of development in the mosquito. Remnants of the mother parasites, consisting mainly of inanimate bodies and pigment, are absorbed by the leukocytes and the vascular endothelium, and thus removed from the circulation. The malarial paroxysms (chill and fever) occur at the time of segmentation (liberation of toxins)—thus at intervals of forty-eight hours (every third day). In some cases a double tertian infection occurs, and segmentation of the different sets of parasites occurring on alternate days, a malarial paroxysm occurs every day (quotidian malarial fever).

The Quartan Parasite.—In general the quartan parasite resembles the tertian, but it presents certain noteworthy differences. In its early stages the quartan parasite is smaller than the tertian and has a more definite contour; its amœboid movements are more sluggish; the pigment granules are coarse, rod-like in shape, and deep brown or blackish in color; and the containing erythrocyte does not increase in size, but remains normal, or perhaps decreases in size, and becomes darker, yellowish, yellowish green, or brassy in color. As the parasite increases in size, the amœboid movement gradually ceases, so that when full-grown (toward the end of seventy-two hours), the parasite is entirely motionless, distinctly outlined, and completely fills the containing erythrocyte. Throughout most of its life cycle the pigment granules are distributed around the periphery of the parasite, but toward the end of seventy-two hours these tend to collect in the centre to form a star-shaped mass radiating from the centre. Segmentation occurs as in the tertian organism, but the segments are more distinctly outlined than in the tertian parasite, they are fewer in number (six to twelve), and they are arranged regularly around the pigment (so-called daisy or marguerite forms). Like the tertian organism, some of the quartan parasites do not undergo segmentation, but persist to undergo the mosquito phase of

development. The malarial paroxysms (chill and fever) occur concurrently with segmentation—about every seventy-two hours (every fourth day). Double or even triple infections with the quartan parasite may occur, giving rise to a malarial paroxysm on each of two succeeding days with a free day, or to a daily paroxysm.

The Estivo-autumnal Parasite.—Two types of the estivo-autumnal parasite may be distinguished, the quotidian and the tertian. The *quotidian estivo-autumnal parasite* is seen in its earliest stages in the infected erythrocyte as a small, round, hyaline or ring-shaped body (signet ring), with indistinct outlines, and markedly amœboid (rapid but not wide movements). The containing erythrocyte is usually smaller than normally, greenish or brassy in color, and sometimes contains two or even three parasites. Some of the parasites become pigmented, in some cases apparently only those in the organs (the spleen and the bone marrow), those in the circulating blood remaining unpigmented; the pigment at first consists of fine, dust-like particles, slightly motile and arranged usually about the periphery; as the parasite grows the pigment tends to collect in the centre and then becomes motionless. When full grown the parasite is scarcely more than one-fourth or one-third the size of a normal erythrocyte (distinction from the ordinary tertian and quartan parasite). Segmentation occurs (in the organs rather than in the circulating blood) at the end of twenty-four hours, and often quite obviously within the erythrocyte; six to eight spores are formed. The *tertian (so-called malignant) estivo-autumnal parasite* differs from the quotidian parasite in that the ring form is larger, more irregular in outline, more sharply defined, and more refractile; it requires forty-eight hours to complete its cycle of development; the pigmented stage lasts twenty-four hours or more, and during this time the parasite is actively amœboid and the pigment exhibits a characteristic oscillatory movement not seen in the quotidian parasite; and when full grown the parasite fills one-half or two-thirds of the infected erythrocyte. This parasite differs from the ordinary tertian organism in being smaller (at corresponding stages of development); in sometimes assuming ring forms and giving rise to crescents; in the more scanty and less active pigment; in occasioning shrinkage (rather than swelling) of the infected erythrocyte; and in giving rise to a less number of spores (eight to fifteen). Segmentation occurs in the internal organs, and often intermittently—whence the irregularity of the clinical manifestations. In both varieties of estivo-autumnal infection, at the end of about a week, other characteristic parasites (with coarse, centrally situated, pigment granules) appear in the blood—crescent-shaped, spindle-shaped (cigar-shaped), and spheroid (or ovoid) bodies. These do not sporulate, but represent sexually differentiated forms (gametocytes) designed for the life cycle of the parasite in the mosquito. The quotidian, as contrasted with the tertian, crescent is smaller, shorter, and plumper; its extremities are rounded (never pointed); its protoplasm is less granular; and its pigment is less in amount. The estivo-autumnal parasites give rise to the more severe

malarial infections, to many latent or masked infections, and to the so-called pernicious or malignant infections, especially common in tropical and subtropical regions; but the ordinary tertian or quartan infections may become pernicious.

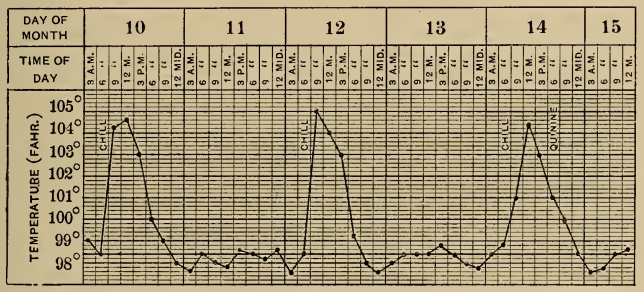
Plasmodium Malariæ in the Mosquito.—When a mosquito of the genus Anopheles bites a malarious subject, it sucks into its stomach blood containing the sexually differentiated forms of the malarial parasite (gametocytes)—large spheroidal non-sporulating forms in the tertian and the quartan infections, and crescents and spheroid (or ovoid) forms in estivo-autumnal infections. These develop into flagellating and non-flagellating spheres, the former representing male elements (microgametocytes) and the latter female elements (macrogametocytes). The flagella (microgametes) are cast off by the male elements, and penetrating fertilize the female body (macrogamete). The impregnated female parasite now penetrates the wall of the midgut of the mosquito and continues its development in the muscular coat, in which, at the end of two days, minute, refractile, and pigmented spheres may be found. These rapidly increase in size and become encapsulated. At the end of a week they may have attained a diameter of 70μ, and they contain a large number of delicate spindle-shaped filaments, about 14μ in length (sporozoites). The mother cell (zygote) soon ruptures, setting free an enormous number of sporozoites, which speedily accumulate in the salivary glands of the mosquito, and are inoculated into man through the proboscis of the mosquito when next it bites. The sporozoites soon develop into young hyaline forms, and, invading the erythrocytes, pass through the asexual cycle of development and give rise to the clinical manifestations of malaria. Fertilization of the macrogamete may also be studied on the warmed stage under the microscope, as was originally observed by W. G. MacCallum.

Pathology.—The most striking lesions in the acute infections consist of destruction of the erythrocytes and certain resultant changes. The blood as a whole is hydremic, the erythrocytes are decreased in number (due to direct destruction and probably also in consequence of a toxin produced by the parasites); the color index is reduced, and granular degeneration and polychromatophilia occur; the mononuclear leukocytes are increased; many of the polynuclear leukocytes are much pigmented; and free pigment is found in the plasma. The spleen is enlarged; the capsule is tense and may rupture; many infected erythrocytes are found in the pulp; and considerable pigment is free and enclosed within macrophages. The liver is enlarged, and hyperemic, and may reveal foci of necrosis due usually to capillary thromboses; the capillaries may contain many parasites. The kidneys are often swollen and may show some cloudy swelling, and may reveal pigmented leukocytes and parasites, acute nephritis is not uncommon in estivo-autumnal infections. The bone marrow often contains many segmenting parasites. In pernicious infections hemoglobinemia and parenchymatous hemorrhages are common, and the capillaries throughout the body (brain, gastro-intestinal

tract, kidneys, etc.) may be found packed with parasites. In chronic malaria there is usually a high-grade anemia; great enlargement, induration, and pigmentation of the spleen (ague cake); enlargement, pigmentation of the endothelial and Kupffer's perivascular cells, and occasionally cirrhosis of the liver; enlargement and pigmentation of the kidneys; pigmentation of the gastro-intestinal mucous membrane, especially of the solitary glands and Peyer's patches; and in the bone marrow replacement of the fat by proliferated marrow cells, new bloodvessels, and pigmentation.

Symptomatology.—The following clinical types of the malarial infections may be distinguished: (1) Tertian infection; (2) quartan infection; (3) estivo-autumnal infections; and (4) chronic malarial cachexia. The tertian and the quartan infections comprise the regularly intermittent fever of the older writers, and the estivo-autumnal infections the irregular, remittent, continued, and pernicious fevers.

FIG. 4



The temperature in tertian malarial infection (Craig).

TERTIAN INFECTION.—Tertian malarial infection is characterized by a so-called malarial paroxysm, consisting of chill, fever, and sweat, recurring at intervals of forty-eight hours (every third day), corresponding with the life cycle of the tertian parasite (Fig. 4). The period of incubation is not definitely known; it is said to vary from thirty-six hours (experimentally) to twenty days. Prodromal symptoms, consisting of malaise, headache, restlessness, yawning, stretching, loss of appetite, etc., usually precede for several days the definite malarial paroxysm. The initial manifestation of the paroxysm, the chill, is, as a rule, sudden in onset, and it is usually severe, the entire body being more or less violently agitated. The skin is pale, cold, cyanosed, and presents the peculiar appearance described as goose-flesh (*cutis anserina*). The patient complains bitterly of the violent agitation and cold, and seeks to mitigate his distress by additional bedclothes, and hot water bottles. His discomfort is sometimes increased by nausea and vomiting and intense headache (congestion of the cerebral vessels). The pulse is frequent, and often hard and

irregular; and the urine may be increased in amount. The chilling of the body is superficial only, since with the advent of the chill (if not previously) the temperature begins to rise, and at the height of the chill the rectal (or the axillary) temperature may be 103° to 106° F. or more. This so-called cold stage lasts for from fifteen minutes to an hour, and is succeeded by the hot stage. The patient begins to complain of alternating sensations of heat and cold, until gradually the subjective sensation of cold disappears and the surface of the body becomes intensely hot, whereupon he complains as much of the heat as he did of the cold. The face is flushed, the conjunctivæ injected, the general integument congested; the pulse becomes full, bounding, and perhaps dicrotic; the respirations are quickened; throbbing headache and thirst are often marked; and in severe infections stupor and delirium may supervene. Cutaneous eruptions—erythema, urticaria, and herpes—often develop, labial herpes being quite as common as in pneumonia. During this stage the temperature reaches its highest point—105° to 106° F. or more, though occasionally it may be only 104° F. The hot stage lasts for from one to three or four hours or more, and ends abruptly with a sudden fall of the temperature to normal (intermittent fever) and a profuse sweat. The perspiration usually begins on the forehead and face and soon involves the entire body, and has been said to have a characteristic sperm-like odor; the pulse becomes slower and weaker; the headache and other subjective complaints cease; considerable urine may be voided; and the patient often falls into a sleep from which he awakens several hours later much refreshed, though often somewhat exhausted. The sweating stage lasts usually from one to three hours.

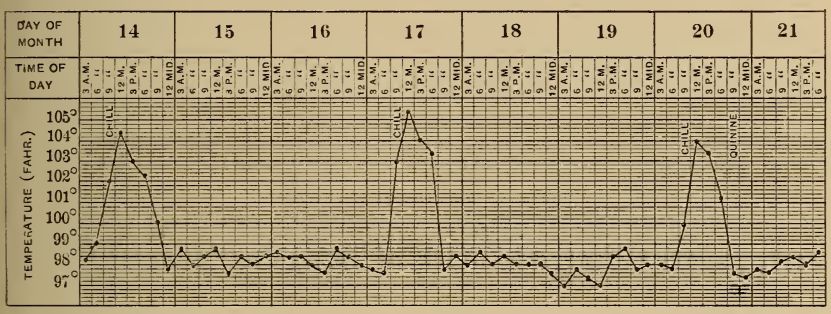
The average duration of the tertian malarial paroxysm is from ten to twelve hours; it may be less when mild, and it is sometimes very severe and much prolonged. One, two, or all three stages may be much shortened or abortive. Frequently, especially in primary attacks and in children, there is not a distinct chill, but only chilliness, or merely pallor and cyanosis in children; or the hot stage alone may be present, the sweating being slight; or the fever may escape observation—whereupon the infection is likely to go unrecognized.

During the paroxysm the spleen becomes enlarged and palpable, and may remain so. The skin may present a peculiar lemon-yellow tint—due to a rapidly developed anemia, a form of hemolytic jaundice, or abnormal deposition of pigment. A dry bronchitis, diarrhœa and other intestinal symptoms, etc., are not uncommon concomitant symptoms, and suggest special localizations of the toxic process.

In some cases the paroxysms occur not every other day, but daily (quotidian intermittent fever): cases of double tertian infection in which the two sets of parasites mature on alternate days. After persisting for several weeks, the malarial paroxysms frequently tend to cease spontaneously, but in this event recurrence is very likely. When neglected the infection may become chronic (malarial cachexia), or rarely pernicious.

Quartan Infection.—Quartan malarial infection is characterized by malarial paroxysms (chill, fever, and sweat) recurring at intervals of seventy-two hours (every fourth day)—corresponding with the life cycle of the quartan parasite (Fig. 5). In the event of a double quartan infection a paroxysm occurs on each of two successive days and is followed by a free day; in the event of a triple infection a paroxysm occurs daily (quotidian fever). As a rule, the paroxysms in quartan infection, as contrasted with tertian infection, are more severe, though they are shorter (usually less than ten hours); nervous symptoms, such as headache, restlessness, etc, are more pronounced; delirium is more likely to occur; and the tendency toward perniciousness is somewhat more marked. Otherwise there is but little difference in the clinical manifestations of the two forms of infection.

Fig. 5



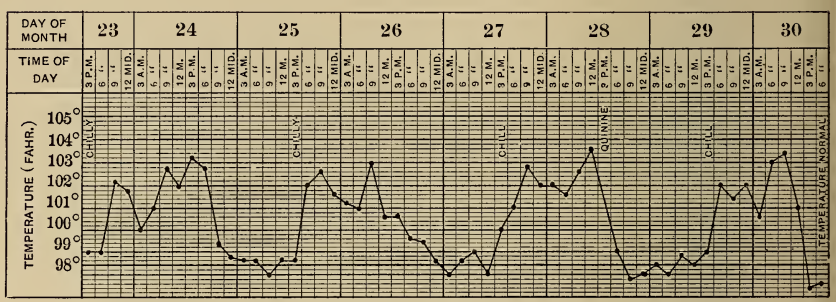
The temperature in quartan malarial infection. (Craig.)

Estivo-autumnal Infections.—These are most common in tropical and subtropical regions, where they prevail throughout the year, but they are also rather prevalent in the temperate zones, especially during July, August, September, and October (whence the name). As a rule, they are more severe than the ordinary tertian and quartan infections, and they are more likely to become pernicious. The following varieties may be differentiated: (1) Tertian estivo-autumnal infection (the most common); (2) quotidian estivo-autumnal infection; (3) pernicious estivo-autumnal infection, of which the comatose, the algid, and the hemorrhagic forms are the most common.

Tertian Estivo-autumnal Infection.—The prodromal symptoms, as a rule, are similar to those of the ordinary tertian and quartan infections. The cold stage does not last more than a half hour, and there is usually chilliness rather than a distinct chill; but the patient complains of headache, pain in the back and legs, nausea, and perhaps vomiting; he is much depressed mentally, and he may be slightly stuporous or even delirious; the skin and mucous membranes are cyanosed; the pulse is frequent, weak, and irregular; the respirations are rapid and shallow;

and the internal temperature is elevated (Fig. 6). At the onset of the hot stage, the temperature has reached 103° to 104° F.; whereupon it oscillates slightly for several hours, then falls a degree or two or three (pseudo-crisis), rises again, perhaps higher than it was previously (precritical rise), and finally falls rapidly to normal or below (true crisis). The entire fever period is much longer than in ordinary tertian infections, and may last twenty, thirty, or even forty hours. Concurrently with the crisis the patient sweats, but the sweating is usually much less marked than in ordinary tertian infections. In many cases the paroxysms recur regularly every forty-eight hours, corresponding with the life cycle of the parasite—a fact not sufficiently recognized; but in many cases the manifestations are quite irregular. The temperature may rise slowly or fall by lysis; and it frequently becomes remittent or continuous in consequence of anticipation (overlapping) or retardation of the paroxysms (especially

Fig. 6



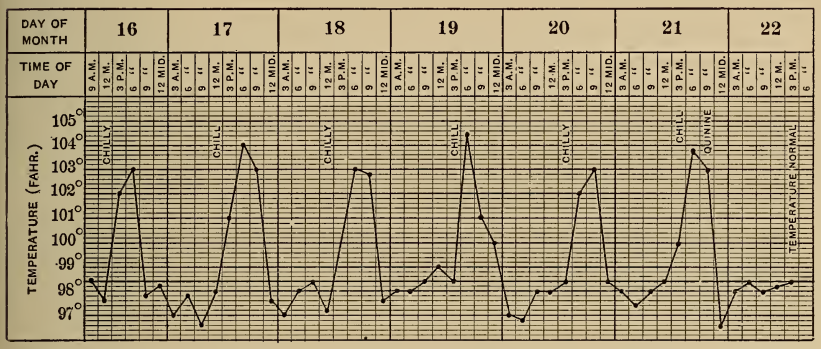
The temperature in tertian estivo-autumnal malarial infection. (Craig.)

common in estivo-autumnal infections), reduplications of the paroxysms due to double or mixed infections, etc.; and sometimes there is a moderate degree of fever between irregularly recurring paroxysms attended by more marked fever. In consequence, the infection has been and frequently is mistaken for typhoid fever: a coated tongue, enlarged spleen, bronchitis, tympanites, and diarrhœa alternating with constipation, increase the diagnostic difficulties—whence the ill-advised term, typho-malarial fever. The patient is depressed, even prostrated, and complains of divers subjective complaints, not only during the paroxysms, but also between them. Frequently the fever subsides at the end of a week or ten days, but it may continue much longer. In severe infections, loss of appetite, nausea, bilious vomiting, and jaundice, suggest the designations bilious remittent fever, gastrobilious fever, jungle fever, etc. These often assume pernicious tendencies.

Quotidian estivo-autumnal infection frequently cannot be differentiated clinically from the tertian estivo-autumnal infection, but, as a rule, it is characterized by more severe chilly sensations or distinct chills and more marked sweating (though neither is as marked as in the ordinary

tertian and quartan infections); the paroxysms recur daily, the fever rising rapidly to 103° or 104° F., and falling suddenly within six or eight hours to normal (Fig. 7); but the paroxysms frequently become prolonged to twelve, sixteen, or twenty-four hours (due to irregular generation of parasites)—whence a remittent or continued fever results (especially in pernicious cases). Postponement of the paroxysms occurs in cases tending to spontaneous recovery.

FIG. 7



The temperature in quotidian estivo-autumnal malarial infection. (Craig.)

Pernicious Estivo-autumnal Infections.—Depending upon the special prominence of certain symptoms, many varieties of pernicious estivo-autumnal infections have been described, such as comatose, delirious, convulsive, syncopal, paralytic, algid, gastro-intestinal, bilious, choleraic, dysenteric, cardialgic, hemorrhagic, hemoglobinuric, pneumonic, etc.; but these represent variations in degree, rather than differences in kind. The so-called comatose, the algid, and the hemorrhagic forms are the most common and merit special mention.

The *comatose* (the most common) form is due usually to the tertian estivo-autumnal parasite, and is characterized by symptoms of intense cerebral congestion (intimately associated with thrombi of parasites in the cerebral capillaries and necrotic and other changes about them). At the onset there is often a chill and fever, as in other malarial infections, though nervousness and mental depression (rarely slight delirium) may be early obtrusive features. Soon the patient becomes stuporous and then comatose. The coma may come on with the first paroxysm, or not until the second or third (or later), and although it may be preceded by mental disturbance, it may develop suddenly, persist, and lead to death within several hours or two or three days. On the other hand, the coma may intermit, that is, develop with the rise of the temperature and clear up when the fever falls—in which event it rarely lasts more than twelve to twenty-four hours; but a second or third attack may prove fatal. The fever is high, but it may be as low as 101° F.; the face is flushed, or

subicteric or leaden, if anemia is present; the pulse is slow and full at first, but it soon becomes weak and irregular; the spleen is enlarged; the pupils may be equally contracted, or unequally contracted or dilated; muscular twitchings are common; and the sphincters may become paralyzed.

The *algid* form (congestive chills) is characterized by nausea, vomiting, and purging, leading to collapse and simulating the algid stage of cholera. Usually there is no chill, but chilliness is complained of; the internal temperature may become considerably or moderately elevated, but it is not infrequently subnormal; the pulse is rapid, weak, and irregular; the general integument and the visible mucous membranes are pale and livid, and the skin is covered with a profuse clammy sweat; the eyes are sunken, the pupils dilated, and the Hippocratic facies develops. The urine is often lessened in amount or suppressed, and jaundice and grave nervous symptoms may supervene (simulating yellow fever). The patient may die within a few hours, or at the end of several days, from profound asthenia.

The *hemorrhagic* form is characterized by bleeding from the mucous membranes of the body (mouth, stomach, intestine, rectum, vagina, etc.), or into the skin and the interstices of the body; but special interest attaches to a peculiar hemorrhagic form of the infection known as blackwater fever, hemoglobinuric fever, or malarial hemoglobinuria. This is rather common in our own Southern States, and in Central America, Italy, and Africa. It usually follows estivo-autumnal infections, but it has been observed after ordinary tertian and quartan infections. Apparently it occurs only in persons who have had repeated attacks of malaria and have become anemic or cachectic. The parasites, as a rule, are present in the blood before the onset of the attack, but during the attack they are often absent. The immediate cause of the attack is not known: those who have seen the disorder are not agreed as to whether or not quinine is of significance in provoking the attack; but there is some evidence that quinine is of etiological importance in some cases, and it often appears to be harmful if administered during the attack when the parasites are absent from the blood (said to be referable to a hemolytic action of the quinine on the erythrocytes). In some cases the patient gives a history of a few days of intermittent or remittent fever with increasing biliousness, then a sudden chill and bloody urine; in other cases, especially those that develop late in the fall or winter, there may have been for some time irregular outbreaks of malaria, then the prodromal symptoms of another attack followed by the bloody urine (said by some observers to be provoked by the quinine taken to ward off the impending paroxysm). The urine contains methemoglobin, hemoglobin, hematin, degenerated erythrocytes, leukocytes, biliary coloring matter, tube casts, and considerable albumin. In favorable cases the urine becomes clear in from three or four hours to three or four days; but the other malarial manifestations may continue. In other cases the hemoglobinuria recurs periodically. Death may occur

on the first day from marked toxemia and coma, or it may occur later from the effects of the blood destruction, suppression of the urine and uremia, and general exhaustion.

Chronic Malarial Cachexia.—This is a condition of pronounced anemia with enlarged spleen that develops in those the subjects of long-continued malarial infection. The symptoms are those common to high grade anemia—asthenia, breathlessness, palpitation of the heart, and slight œdema about the ankles. In addition there is a subicteric hue to the skin or a yellowish-brown pigmentation; the spleen is enlarged; hemorrhages may occur into the retina and elsewhere (especially the gastro-intestinal tract); and the patient is subject to irregular attacks of fever of variable duration with intervening periods of apyrexia.

Latent and Masked Malarial Infections.—These are by no means uncommon, and though usually due to the estivo-autumnal parasite, they may be due to the ordinary tertian or quartan parasite. These infections, though they often manifest a noteworthy periodicity, simulate a wide variety of disorders, such as neuralgia (supra-orbital, occipital, intercostal, sciatica, etc.), paresthesias, anesthesias, paralyses, hemiplegia, ataxia, amaurosis, deafness, etc., and certain gastro-intestinal disorders, such as gastro-enteritis, dysentery, etc. In many of the cases the parasite may be found in the blood; in other cases, the symptoms subside promptly upon the administration of quinine. Tuberculosis, dysentery, cardiac degeneration, cirrhosis of the liver, nephritis, orchitis, parotitis, gangrene of the skin, etc., have been observed as complications or sequels of malarial infections.

Diagnosis.—The ordinary tertian and quartan infections are usually recognizable from the clinical manifestations, but the diagnosis is easily rendered certain by a microscopic examination of the blood. This should not be neglected in any case in the diagnosis of which malaria enters as a possibility. In the event of more continued fever, it may be necessary to exclude typhoid fever, tuberculosis, pyogenic infections (especially acute ulcerative endocarditis), etc. Typhoid fever may be excluded by the absence of the Gruber-Widal reaction, the presence of malarial parasites in the blood, and the response of the symptoms to quinine; in fact, that fever that does not respond to quinine within four or five days may be said not to be malaria. Tuberculosis may be excluded by the history of the patient, the associated general symptoms, the physical signs, the presence of tubercle bacilli in the sputum or cerebrospinal fluid (or choroid tubercles), and the absence of malarial parasites in the blood, and the non-response of the symptoms to quinine. Most of the pyogenic infections, of which ulcerative endocarditis is a type, are attended by leukocytosis (which is absent in malaria), they do not respond to quinine, and malarial parasites are not found in the blood.

Prognosis.—In the ordinary tertian and quartan infections the prognosis is good; the disorders are quite amenable to treatment. The prognosis is also relatively good in many of the latent and masked infections, though they may last a long time. But the outlook is very bad

in many of the estivo-autumnal, especially the pernicious, infections—the mortality being very high.

Treatment.—*Prophylaxis.*—The malarial infections may be prevented by protecting all persons from the bites of mosquitoes. In malarious districts the windows and doors of all houses should be screened, and additional protection should be afforded by surrounding the beds with mosquito netting, especially the beds of malarious subjects (to prevent infection of uninfected mosquitoes). Furthermore, the breeding places of mosquitoes should be destroyed by filling up and draining swamps, and preventing stagnation of surface water. Larvæ should be destroyed by coating the surface of all stagnant pools with kerosene (one ounce to each fifteen square feet of water)—whereby the larvæ are deprived of oxygen; this should be repeated every three or four weeks. Small fish also are of service in destroying the larvæ. Oil of pennyroyal or of eucalyptus (which is obnoxious to mosquitoes) may be smeared on the hands by those obliged to work out of doors in malarious districts. Five to ten grains (0.3 to 0.6 gram) of quinine, once or twice a week, is also of service in warding off malarial attacks.

Quinine is a specific remedy and the only drug that really can be depended upon. Following ancient custom, it is wise to deplete the liver and to unload the bowels by a preliminary dose of calomel (10 grains, 0.6 gram), followed by a saline. Since the quinine is most efficacious when the parasites are free in the blood plasma, rather than when they are within the erythrocytes, it is desirable to have an adequate amount of quinine in the circulation at the time of sporulation. Therefore, four to six hours before the expected paroxysms in tertian and quartan infections, 15 to 30 grains (1 to 2 grams) of quinine bisulphate or bihydrochlorate should be given in solution (or in capsule or cachet, but not in pill form, since the pills often do not dissolve). If administered only shortly before the paroxysm the quinine will not prevent it, but will exert a deleterious effect on the parasites and prevent a recurrence. After the initial dose, it is wise to continue the quinine in from 10 to 20 grain (0.6 to 1.3 gram) doses for several days, and then in 5 grain (0.3 gram) doses daily for a week, and thereafter at intervals of a week for a month. In estivo-autumnal infections, similar doses, as a rule, are effectual, but in severe infections larger doses (up to 60 grains, 4 grams, or more, daily) may be required. In pernicious types of the infection it is often necessary to bring the system rapidly under the effects of the quinine, in which event it should be administered hypodermicly or intravenously: 15 to 30 grains (1 to 2 grams) of the bihydrochlorate, or of the hydrochlorate of quinine and urea, in 2 drams (30 c.c.) or more of physiological saline solution; if necessary, this may be repeated every two to four hours. During the attack of blackwater fever, quinine should not be administered, but it may be given in the intervals if parasites are found in the blood. No really efficient substitute for quinine has been discovered; methylene blue is a poor substitute, but may be given in doses of 1 to 4 grains (0.06 to 0.25 gram) when quinine cannot be taken.

During the malarial paroxysms the patient should be made as comfortable as possible—hot water bags during the cold stage, and tepid sponging and cooling drinks during the hot stage. Unduly severe paroxysms should be controlled by an opiate. If necessary, the circulation should be supported by stimulants—strychnine and alcohol. Vomiting may be controlled by counterirritation to the epigastrium, cracked ice, sips of hot water, spirit of chloroform, a small amount of dry champagne, cerium oxalate and bismuth, etc.

Many latent and masked infections are immediately relieved by one or two large doses of quinine; others require the prolonged use of the drug. Warburg's tincture is sometimes efficacious. In chronic malarial cachexia, removal to a non-malarious region, preferably at a high altitude, arsenic (Fowler's solution or arsenous acid), small doses of quinine and iron, or Warburg's tincture will be found efficacious.

METAZOAN INFECTIONS.

DISTOMATOSIS.

(*Distomiasis; Trematode Infections; Fluke Infections.*)

In man there are four important clinical forms of distomatosis—pulmonary, hepatic, intestinal, and venal or hemal distomatosis.

Pulmonary Distomatosis (Lung-fluke Disease); Paragonimiasis; Parasitic Hemoptysis.—Pulmonary distomatosis occurs principally in Japan and China, but also in the United States, the Philippines, and Formosa. The parasite, Paragonimus (Distoma) westermanii, is an oval spinose fluke, somewhat pinkish in color, and measuring from 7.5 to 16 mm. long, 4 to 8 mm. broad, and 2 to 5 mm. thick. The original host has not been determined with certainty, but the organism has been found in man, cats, dogs, swine, etc. The source of infection in man is not known, but it is probably contaminated food or water. The adult parasites are found in cysts in the lungs, pleura, brain, liver, abdominal cavity, etc. The symptoms consist of cough, yellowish, reddish, or rusty-brown sputum, in which ova (smooth, oval organisms, 80 to 100 μ in length, by 40 to 60 μ in breadth) may be found, often in great abundance (12,000 daily). In some cases attacks of more or less severe hemoptysis occur. The liver, brain, eyelids, etc., may become involved, and death not infrequently results.

Hepatic Distomatosis (Liver-fluke Disease.).—Six different species of flukes have been found in connection with hepatic distomatosis: (1) Opisthorchis sinensis (Distoma sinense), the most important, the cause of Asiatic opisthorchiasis or Japanese liver-fluke disease. The organism is an elongate, lanceolate, non-spinose trematode, 9.7 to 20 mm. long and 2 to 5 mm. broad; it is of a reddish color and very transparent. Its chief

anatomical characteristic is the branched condition of the testicles. The eggs are dark brown in color, have a well-defined operculum, and measure 24 to 30 by 15 to 17.5 μ. Man, cats, and dogs appear to be normal hosts. Infection seems to be acquired from swallowing the free cercaria or indirectly from eating an infected water animal (snail, fish, etc.) or food contaminated by infected water. (2) Opisthorchis felineus, the cause of Siberian opisthorchiasis or Siberian liver-fluke disease. Cases in man have been reported in Siberia and Prussia. The organism is a lanceolate, non-spinous trematode, 8 to 18 mm. long and 1.25 to 2.5 mm. broad; the testicles are lobate, but not branched. The eggs are yellowish brown, oval, have a well-defined operculum, and one side flatter than the other, and measure 26 to 30 by 11 to 15 μ. The cat, dog, fox, and glutton are natural hosts. Infection seems to be acquired from eating raw fish. (3) Opisthorchis noverca, the cause of Indian opisthorchiasis or Indian liver-fluke disease, which is very rare. (4) Dicrocœlium lanceatum, the cause of dicrocœliasis (lancet-fluke infection). The organism is a lancet-shaped trematode, 4 to 9 mm. long and 2 to 4 mm. broad. The eggs are dark brown and thick shelled, have a distinct operculum, and measure 38 to 45 by 20 to 30 μ. Cattle and sheep are normal hosts. The infection is rare in man, and probably always accidental. (5) Fasciola hepatica, the cause of fascioliasis. The organism is a broad flattened, oval trematode, 18 to 30 mm. long and 4 to 13 mm. broad. The eggs are yellowish or brownish in color, 130 to 145 by 70 to 90 μ, and they have a distinct operculum. Cattle, goats, and sheep are normal hosts; snails are the intermediate host. Infection is acquired by swallowing the encysted cercaria. The infection is rare in man. (6) Fasciola hepatica, or an organism allied thereto, is said to be the cause of a disease known as halzoun in Liban, Syria. These liver flukes enter the bile ducts and cause (mechanically and by irritation) stoppage of these passages. In early cases the liver is enlarged and hyperemic; in protracted cases it may be normal or lessened in size, and there may be some sclerosis. The *symptoms* consist principally of enlargement and tenderness of the liver, preceded, attended, or followed by diarrhœa (irregular and intermittent), with dark, sometimes bloody stools, occasionally slight fever (100°). Night blindness, intermittent ascites, anasarca, and epistaxis may occur. Anemia and emaciation supervene, and finally, after more or less improvement and relapses (diarrhœa, etc.), the patient usually dies. The ova are readily found in the stools. Preventive measures comprise destruction of infected animals and their excreta, the use in infected localities of boiled water and thoroughly cooked food, and removal from infected regions. There is no efficient treatment.

Intestinal Distomatosis (Intestinal Fluke Disease).—Intestinal distomatosis in man may be caused by: (1) Fasciolopsis buskii, the largest and most important; it measures 27 to 37 mm. in length, 5.5 to 14 mm. in breadth, and 1.5 to 2 mm. in thickness. It occurs especially in Asia, and is distinguishable from Fasciola hepatica by its large acetabulum

and simple intestine. Infection is said to cause indigestion, nausea, headache, and diarrhœa with bloody stools. The ova which may be found in the stools measure 120 to 130 by 77 to 80 μ. Thymol and calomel are said to be capable of expelling the parasite. (2) Fasciolopsis rathonisi (reported once in Asia). (3) Heterophyes heterophyes, a minute harmless trematode found in the small intestine of man, dogs, and cats. (4) Gastrodiscus hominis (found in the cecum and colon of man in India). (5) Cladorchis (Watsonius) watsoni (reported once in a negro boy in Africa).

Venal Distomatosis (Blood-fluke Disease, Bilharziosis; Egyptian Hematuria; Endemic Hematuria; Bilharzian Hematuria; Bilharzia Disease).—Blood-fluke infection in man may be due to: (1) Schistosoma hæmatobium (Bilharz), the African blood fluke, of which the male is 4 to 15 mm. long and 1 mm. broad, and the female (carried in the gynecophoric canal of the male) longer and filiform. The eggs are 135 to 160 by 55 to 66 μ, provided with a terminal spine, but no operculum. (2) Schistosoma japonicum (Katsurda), the Asiatic blood fluke, of which the male is 7 to 12 mm. long, and 0.53 to 0.8 mm. broad; the female is 8 to 12 mm. long. The eggs are 60 to 90 by 30 to 50 μ, and are not provided with a terminal spine or an operculum. The source of infection is not definitely known, although there is increasing evidence that it occurs through the skin; drinking water cannot be eliminated; some observers believe that the urethra and the anus may be the infectious atria. The young worms live in the veins of the liver; the eggs are deposited in various organs (liver, intestine, bladder), and escape from the body with the urine and feces. One-half of the people of Egypt are believed to be infected; the males more than the females, and the rural rather than the urban population. The *symptoms*, (usually preceded by a period of incubation of four or five months) may be chiefly urogenital, consisting of irritability of the bladder, hematuria, pains in the lumbar region, left iliac fossa, thigh, or vulva, either spontaneous or on micturition; or they may be chiefly rectal, consisting of bloody stools, diarrhœa, and perhaps prolapse of the rectum. Secondarily cystitis, vesical calculus, urinary fistulas, nephritis, vaginal and rectal growths may develop. The patient becomes anemic and debilitated, and after a year or two may die, often from some intercurrent infection. The *diagnosis* is readily made by examining the urine and feces for the ova; in some cases it may be necessary to examine scrapings from the bladder or the rectal wall to detect the parasites. The *prognosis* in mild infections is favorable, although relapses are common. The *treatment* is largely symptomatic and palliative. Good results are said to follow the use of male fern (15 grains, 1 gram, thrice daily), methylene blue (3 grains, 0.2 gram, thrice daily), santonin, quinine, etc. In rectal infections, local sedatives, as in dysentery, often mitigate the suffering.

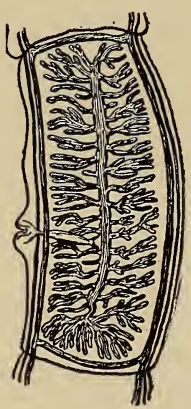
TENIASIS.

(*Cestode Infection; Hydatid Disease.*)

Teniasis may occur in one of two forms: (1) Intestinal infection with the adult cestode—tapeworm; and (2) somatic infection with the larval cestode.

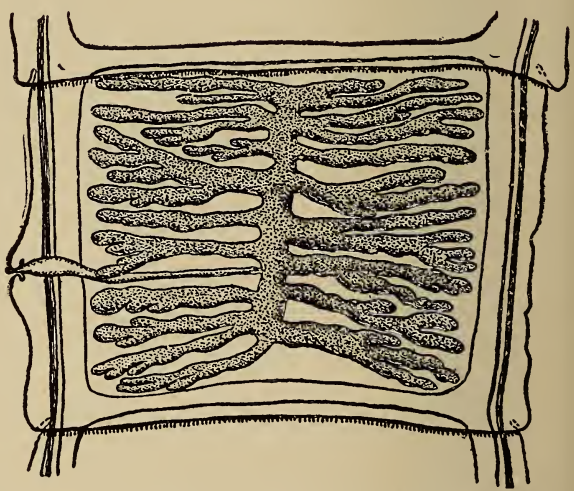
Intestinal Teniasis (Tapeworm Infection).—Stiles states that, so far as established, man seems to be the normal and sole host for the sexual stage of at least two large tapeworms, Tænia saginata and Tænia solium. Man, together with the dog, probably forms the normal host for a third large tapeworm, Dibothriocephalus latus. In common with rats and mice, man seems to have become a normal host for the dwarf tapeworm, Hymenolepis nana, although it has not been definitely established that the worm in man is exactly identical with that of the rodents. Occasionally, though rarely, as accidental host, man harbors the double-pored tapeworm, Digylidium caninum, of dogs and cats, the flavopunctate tapeworm, Hymenolepis diminuta, of rats, and the lanceolate tapeworm, Hymenolepis lanceolata, of ducks and geese. Other rarer examples of tapeworm infection in man have also been reported.

FIG. 8



Gravid segment of Tænia saginata, showing the lateral branches of the uterus, enlarged. (Stiles.)

FIG. 9

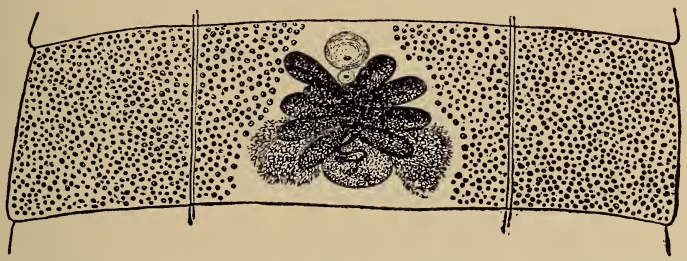


Gravid segment of Tænia solium, showing the lateral branches of the uterus, enlarged. (Stiles.)

Tænia saginata (*mediocanellata*) the fat, unarmed, or beef (beef-measle) tapeworm, is the commonest tapeworm in North America. It may attain a length of 4 to 10 meters; the head, which may measure

2 mm. in breadth, is larger than that of Tænia solium, it is square-shaped, and has four large sucking disks, but no hooklets. There may be as many as a thousand segments; only two ovaries are found in each mature segment; the uterus in the gravid segments has 15 to 35 slender dichotomous lateral branches each side of and shorter than the median stem (Fig. 8); genital pores lateral (marginal), irregularly alternate; terminal segments attain a length of 16 to 25 mm., and a breadth of 4 to 7 mm.; the eggs are provided with a thick, dark, radially striated embryophore (so-called shell), and measure 30 to 40 by 20 to 30 μ. Cattle become infected by swallowing the embryo with food or water which has become contaminated by infected human feces. Man acquires the infection by eating beef, especially the tongue and the muscles of mastication, infected with Cysticercus bovis.

Fig. 10



Gravid segment of Dibothriocephalus latus, showing the rosette uterus in the median line. × 6. (Leuckart.)

Tænia solium, the armed or pork-measly tapeworm, is most common in Europe and Asia. Usually it does not attain a length of more than 2 to 3.5, sometimes 6 to 8 meters; the head is armed with a rostellum bearing a double row of hooks, of larger and smaller size, 22 to 32 in number; genital pores lateral (marginal) irregularly alternate; mature segments contain three ovaries, due to the fact that the ovary on the pore-side of the segment is divided; the segments may attain 10 to 12 mm. in length by 5 to 6 mm. in breadth; 800 to 900 segments may be present; in gravid segments, the median uterine stem possesses 7 to 15 lateral dichotomous branches on each side (Fig. 9); "eggs" (embryophore) are very similar to those of Tænia saginata, and measure 31 to 36 μ in diameter. Hogs become infected by eating infected human feces; and man becomes infected by eating undercooked, underpickled, or undercured pork or wild boar meat, infected with Cysticercus cellulosæ.

Dibothriocephalus latus, the broad, or fish tapeworm, is cosmopolitan in its geographical distribution, but is most common in the vicinity of large bodies of water. It attains a length of 2 to 9 or 10 meters, and a maximum breadth of 20 mm.; it is usually grayish yellow to brownish in color, and composed of 3000 to 4200 segments, which are usually broader than long, especially in the anterior two-thirds of the strobila; the posterior segments become quadrate or even longer than broad, and

are especially characterized by the rosette spot (uterus) in the centre (Fig. 10); the genital pores are ventromedian; the eggs measure 68 to 71 by 45 μ, have a distinct operculum, and are laid during segmentation. Infection in man is acquired by eating raw or underdone fish, especially pike, ling or turbot, perch, and salmon, in the muscles and organs of which the larval stage of the parasite (a "plerocercoid") may be found.

Hymenolepsis nana, the dwarf tapeworm, the smallest tapeworm known for man, is more or less cosmopolitan in its geographical distribution; it is 5 to 45 mm. long and 0.5 to 0.9 mm. in maximum breadth, and is composed of 100 to 200 segments; the rostellum is armed; there are three testicles to each segment; the genital pores are unilateral; the eggs have two distinct membranes, the outer, 30 to 60 μ in diameter, and the inner, 16 to 34 μ in diameter, and present at each pole a more or less conspicuous mammillate projection provided with filamentous appendages. The source of infection in man is not definitely known, but there is reason to believe that it occurs from food infected by the excrement of rats and mice infected with Hymenolepsis nana fraterna (Stiles). Stiles believes that autoinfection is highly probable.

Symptoms.—The symptoms of teniasis are quite variable, depending upon the species of tapeworm present, the number of parasites, and the age and general and nervous condition of the person infected. Often there are no symptoms, the parasite being an accidental finding in the stools. The appetite is variable; salivation may occur; nausea, vomiting, and a feeling of faintness may be present. Abdominal pains are common. They may be colicky or gastralgic in character, of varying intensity, and may be referred to different parts of the body. They may be intermittent, and may be increased by certain foods (herring, onions, and sour food), or decreased by a diet of milk, eggs, and oily food. Anemia is usually not marked in Tænia solium infection; it is more marked in Tænia saginata infection, and still more marked in Dibothriocephalus latus infection. Unequal pupils, disorders of vision, headache, tinnitus aurium, itching and dryness of the nose, epistaxis, and puruitus may be observed. Emaciation and loss of strength sometimes supervene in cases of long standing.

Diagnosis.—The diagnosis is readily made from finding segments of the parasite in the stools, in the absence of which the disease at the most can be merely suspected.

Treatment.—Prophylaxis is of the greatest importance; all meat at abattoirs should be carefully inspected, and that used for food should always be thoroughly cooked (which kills the parasites); infected stools also should be subject to incineration. With a view to cause expulsion of the tapeworm, the patient for a day or two should have the bowels thoroughly evacuated by means of saline cathartics; he should eat very little (mostly fluids), and from about six o'clock in the evening nothing whatever, this with a view to uncover the parasite, permit ready access thereto by the vermifuge, and favor relinquishment of the hold of the parasite to the mucous membrane. In the morning one may give

1 to 2 drams (4 to 8 grams) of the oleoresin or the ethereal extract of male fern, and in about two hours a saline cathartic (not castor oil—which leads to absorption of the male fern and toxic symptoms); to an emulsion of the male fern one may add 2 minims (0.12 gram) of croton oil. Or one may use a decoction of pomegranate root: three ounces (100 grams) of the bark to 10 ounces (300 grams) of water, should be reduced to one half this amount by evaporation, and this taken in divided doses within two to three hours. The active principle pelletierine may be given in doses of 6 to 10 grains (0.4 to 0.6 gram); it also should be followed in an hour by a saline cathartic. Decorticated pumpkin seed, kooso, kamala, santonin, thymol, etc., have also been recommended. Often a large part of the worm is brought away, but the head remains; whereupon the worm continues to grow. Attempts to remove it should be made again at the end of a month or two, when segments again appear in the stools.

Somatic Teniasis.—Infection with the larval stage of tapeworms may be due to: (1) Echinococcus granulosa (hydatid disease); (2) Cysticercus cellulosæ, the larval Tænia solium; and (3) Sparganum mansoni and Sparganum proliferum.

Echinococcus granulosa (*Tænia echinococcus*) is one of the smallest tapeworms; it is composed of a head with twenty-eight to fifty hooks, a short neck, and three or four segments; the first segment is immature, the second is mature, and the last is gravid and composes about one half of the total length (2.5 to 5 mm.) of the worm. The larval stage is the largest cestode known—the echinococcic hydatid; a subspecies is known as Echincoccus multilocularis. The gravid terminal segment of the tapeworm is discharged in the feces of the dog, and the egg gains access to the intermediate host (sheep, cattle, hogs, man, etc.) through contaminated food or drinking water, or, in the case of man, possibly also from hands soiled while petting dogs. Upon arriving in the stomach. the oncosphere (six-hooked embryo) escapes from the shell and, by means of its hooks, bores its way to various parts of the body, especially to the liver. Here it comes to rest, and increasing gradually in size it presents a thick outer cuticle and an inner parenchymatic layer surrounding a cavity containing fluid. An outer connective tissue cyst is furnished by the host. Brood capsules arise from the parenchymatic layer and hang into the cavity; heads form in these brood capsules. Daughter and even granddaughter cysts may form and fall into the cavity of the mother cyst (so-called, endogenous echinococcus, common in man), or these daughter cysts may pass outside the mother cyst in the surrounding tissue (exogenous echinococcus, uncommon in man). Iceland is the home of the echinococcic infection, but it occurs in all parts of the world, although it is rather rare in this country. The infection is most common in the liver (56 per cent.), in the lung (8 per cent.), and in the kidneys (7 per cent.); but it may occur in any part of the body.

Symptoms.—The symptoms consist of a slowly growing tumor, which may attain very large dimensions. The tumor may be more or less elastic, and may manifest on percussion a so-called hydatid thrill or

fremitus. The nature of the growth may be revealed by detecting hooklets in the fluid. Sugar in the fluid also is suggestive. A Wassermann and a precipitin-reaction test have been suggested.

Treatment.—The treatment is surgical.

Cysticercus cellulosæ, the larval form of Tænia solium, is an elliptical translucid, bladder-like structure, 6 to 12 mm. long by 5 to 10 mm. broad, with a white spot at its equator, due to the invaginated head. The disorder to which it gives rise, cysticercosis, is characterized by the presence of the parasite in the subcutaneous tissues, muscles, brain, spinal canal, eye, heart, lymph nodes, etc. It takes about three months for the parasite to develop from the six-hooked embryo (onchosphere) to the bladder worm stage, which may live in man as long as twenty years. The *symptoms* vary with the location and the number of parasites (sometimes several thousand). In slight infections there may be no symptoms; in severe infections, involving the brain there may be headache, visual disturbances (ophthalmoscopic examination may reveal the parasite), mental disorders, dizziness, paralyses, convulsions, etc. There is no effective medical treatment, although some good results with male fern have been reported. From accessible regions (the eye, for instance), the parasite may be removed surgically.

Sparganum mansoni, a larval bothriocephalide tapeworm, has been rarely found in man (Japanese) in the peritoneal cavity, eye, urethra, etc. *Sparganum proliferum* has been reported once.

NEMATHELMINTHIASIS.

(*Roundworm Infection.*)

Uncinariasis (Ankylostomiasis; Hookworm Disease; Miner's Anemia; Egpytian Chlorosis).—Uncinariasis is common in tropical and subtropical countries. It is estimated that 90 per cent. of the rural population of Florida, and 30 to 80 per cent. of the miners of Germany are infected; but the disorder occurs also in miners and others who are close in contact with the soil in more temperate zones. There are two parasites: (1) Necator americanus (Uncinaria americana, Stiles), the New World hookworm, a cylindrical worm, 5 to 11 mm. long, and possessed of a dorsal and a ventral pair of lips at the mouth, a prominent dorsomedian buccal tooth, and four buccal lancets; in the male, the dorsal ray of the bursa divides at the base and each branch possesses two tips; in the female the vulva is the anterior half of the body; the eggs are thin-shelled, 64 to 72 μ long, by 36 to 40 μ broad; they are oval with somewhat bluntly rounded poles. (2) Ankylostoma duodenale (Uncinaria duodenalis, Dubini), the Old World hookworm, is 8 to 18 mm. in length and possesses in its mouth two pairs of strong, curved, ventral teeth, and one pair of knob-like dorsal teeth; the dorsomedian tooth of the buccal capsule is nil or practically so; a pair of ventral lancets is present in the buccal cavity; in the male, the dorsal ray of the bursa is

divided two-thirds its length from the base, and each branch is subdivided into three tips; in the female the vulva is in the caudal half of the body; the eggs measure 52 to 61 μ in length and 42 to 38 μ in breadth; they are oval and have bluntly rounded poles. Both parasites inhabit the small intestine, especially the jejunum and ileum, but also the duodenum and occasionally the stomach. Infection may occur (1) through the skin, whence the larvæ reach the circulatory system and eventually gain the small intestine; and (2) by way of the digestive tract with contaminated food or water or dirt on the hands. The eggs are oviposited in the intestine of the patient, but do not develop until after they escape with the feces. The conspicuous lesions consist of general anemia, deficient coagulability of the blood (due to a toxin produced by the hookworm), and catarrh of the intestinal tract.

Symptoms.—The symptoms vary with the severity of the infection. Ova may be present in the stools without noteworthy symptoms. The chief manifestations consist of gastro-intestinal irritability, such as poor appetite, nausea, vomiting, epigastric distress, flatulence, etc.; slight fever; and in advanced cases secondary anemia, with high leukocytosis and eosinophilia. The patient tires readily and complains of lassitude. The liver and spleen may become enlarged; the heart may become weak and dilated, and œdema may develop. The diagnosis is readily made by finding the worms or the ova in the stools. The outlook is favorable in early cases, but the mortality is about 30 per cent.

Treatment.—Efforts should be made to minimize and prevent the infection by insisting upon the wearing of shoes in infected regions, examining the stools of persons in infected regions, and carefully destroying contaminated stools, and disinfecting privies. The patient should be put on a fluid diet for a day; in the evening he should take a saline cathartic, but no food. In the morning an adult should be given 30 to 45 grains (2 to 3 grams) of thymol (in cachets with an equal amount of milk sugar), and at the end of three or four hours (earlier if symptoms of intoxication develop), he should take another saline cathartic. This may be repeated at the end of a week if worms or ova are still present. Beta-naphthol and male fern also have been recommended. The anemia requires the use of iron.

Strongyloidosis.—Infection with Strongyloides stercoralis is common in tropical and subtropical zones (75 per cent. of the anemias of India); but it occurs also elsewhere, including the United States. Infection may occur through the skin, or by means of contaminated food or water. The parasite apparently may be harmless, or it may set up an intestinal catarrh and diarrhœa. The diagnosis may be made by finding the ova in the stools. Thymol, as in uncinariasis, is the best treatment, but it is not always efficacious, since the parasite may burrow into the tissues.

Ascariasis (Eelworm Infection).—Infection with Ascaris lumbricoides (Linnæus) is widespread, but is most common in warm climates. It may occur at any age, but is most frequent in children. Several or many worms may be present in the one case. The male parasite

measures 15 to 17 even 25 cm. in length and about 3 mm. in diameter; the female, 20 to 40 cm. in length and 5 cm. in diameter, and is oviparous; they are grayish to reddish yellow in color; the anterior end is provided with three lips; the eggs are 50 to 75 by 36 to 55 μ, unsegmented when oviposited, and provided with a thick mamillate covering, frequently tinged yellow when found in the feces. The parasites live in the small intestine. Development is direct, without an intermediate host. Infection is acquired by contaminated drink, fruit, hands, etc.; the fly may be an active agent in transmitting the infection. The embryo being swallowed develops directly into the adult worm. *Symptoms* may be entirely in abeyance, or there may be various ill-defined gastro-intestinal manifestations, or nervous symptoms in children, and eosinophilia. The disorder, of course, is recognized when a worm is passed by the rectum or is vomited. In other cases the ova may be found in the stools. The best *treatment* consists of the use of santonin, $\frac{1}{2}$ to 1 grain (0.03 to. 0650 gram) to a child, 2 to 3 grains (0.13 to 0.20 gram) to an adult, with or followed by calomel, and later a saline cathartic. This may be repeated on the third and the fifth day. Thymol and oil of chenopodium also have been recommended.

Oxyuriasis (Pinworm Infection; Threadworm Infection; Seatworm Infection).—Infection with Oxyuris (Oxyurias) vermicularis (Linnæus) is widespread, and very common in children and women. Few or many worms may be present in the one individual. The parasite is small, white, and round, and measures 3 to 5 mm. (male) to 10 mm. (female) in length and 0.16 to 0.6 mm. in diameter; the male has but one spicule; the female is provided with a relatively long, sharply pointed tail; the vulva is in the latter half of the anterior of the body; two uteri are present filled with numerous eggs; these eggs are 50 to 52 by 16 to 24 μ, and have a thin shell and a dorsal surface much more convex than the ventral. The fertilized females wander to the cecum, and later when gravid to the colon. Infection is acquired by auto-infection (infecting the finger nails by scratching the anus to relieve irritation occasioned by worms wandering from the rectum—the worm is thus often crushed); from a bed-fellow, or a nurse, or the bedclothes; or from infected food or drink. Flies possibly transmit the infection to food, etc. Development is direct —without an intermediate host. *Symptoms* may be entirely in abeyance —in slight infections. In severe or heavy infections, gastro-intestinal symptoms are common, especially foul breath, abdominal distress, nausea, vomiting, diarrhœa, etc., as well as nervous symptoms, such as headache, restlessness, etc. The anus is often markedly irritated, and itches and burns, from out-wandering of the female parasite; these may invade the vagina, even the uterus and the peritoneal cavity. The *diagnosis* is readily made from detecting the worms in the stools or about the anus, or from finding the ova in the stools. The best *treatment* consists of santonin and calomel, as mentioned under ascariasis. Thymol, beta-naphthol, and a bitter infusion, such as gentian, have also been recommended. In addition, with a view to rid the rectum of worms, one

should use daily, for a week or ten days, enemas of an infusion of quassia, or turpentine and water, or saline solution, etc. The itching about the anus may be relieved by belladonna or zinc oxide ointment.

Trichocephaliasis (**Whipworm Infection**).—Infection with Trichuris trichiuria (Linnæus) is widespread, and occurs in man, apes, etc. The parasite has the general form of a whip; the male measures 40 to 45 mm. and the female 45 to 50 mm. in length; the eggs measure 50 to 54 by 21 to 23 μ, and are of a yellowish or dark brown color, with unsegmented protoplasm and a peculiar light spot at each pole resembling apertures. The parasites inhabit the cecum, but may be found in the appendix, colon, and ileum. *Symptoms,* as a rule, are entirely in abeyance. In some cases a more or less well-marked secondary anemia results. Appendicitis is said to have been provoked by the worms, which also have been credited with transporting infecting bacteria (typhoid bacilli). The *diagnosis* is readily made by finding the worms or the eggs in the feces. The *treatment* is unsatisfactory: thymol and male fern have been recommended, as well as benzine internally and irrigations of the colon with benzine, 1 dram (4 c.c.) to a quart (liter) of warm water.

Acanthocephaliasis.—Infection with Acanthocephali (thorn-headed worms) is of very little importance in human medicine.

Trichinosis (**Trichiniasis**).—Infection with Trichinella spiralis is widespread, and is found in man, hogs, wild boars, rats, dogs, and cats. Stiles states that three stages of the parasite should be clearly held in mind: (*a*) The adults live in the duodenum and jejunum; the males measure 1.4 to 1.6 mm. in length by 40 μ in diameter, while the females are 3 to 4 mm. long by 60 μ thick; they are circular on cross section, and appear as minute thread-like objects; the œsophagis is supported by a single row of cells known as the cell-body; the male is without spicules; the female is viviparous, the vulva being situated about one-fifth the length of the body from the mouth. The males die shortly after copulation. The females may remain for a few weeks in the lumen of the intestine, or they bore into the lymphatic spaces of the intestine where they live about five to seven weeks and deposit their numerous young, about 1500 or more per female; (*b*) embryos, which measure about 90 to 100 μ in length by 6 μ in breadth; these wander, either with the lymph or with the blood, less frequently actively, to the striated muscles; they begin to reach the muscles about the tenth day after infection; they enter the muscle fibers and there develop into (*c*) encysted larvæ; these cysts vary somewhat in size, but are usually about 400 by 250 μ. The encysted larvæ may remain alive in the muscles for years, cases being reported for as long periods as twenty to thirty-one years. The encysted worm (the "fleshworm") is the infecting stage, found in the hog; upon being swallowed in raw or rare pork, the cyst is destroyed, the larvæ pass from the stomach to the small intestine and develop within about two days or less to the adults; the latter copulate and may have embryos in the uterus within less than a week after infection. Man becomes infected

from eating pork; hogs, from eating uncooked swill containing scraps of pork, or swine offal at country slaughter houses, or rats; and rats, from eating one another, scraps of pork in houses or meat-shops, and swine offal at country slaughter houses.

Symptoms.—The symptoms may be slight and escape observation. Typical cases present Rupprecht's three more or less well defined periods, corresponding to the three stages of the parasite and their respective location: (1) The period of ingression, marked by predominance of gastro-intestinal symptoms—anorexia, nausea, vomiting, diarrhœa (usually profuse), sometimes succeeded by constipation, recurrent abdominal pains, vague pains in the muscles, a primary and transient œdema of the eyelids (about the eighth day), etc. (2) The period of digression, marked often by rather abrupt onset about the ninth to the fourteenth day (rarely later), fever, pain and tenderness of the muscles (especially the biceps and the gastrocnemius), which become firm and resistent, especially on motion; this, as a rule, is exceedingly painful, and may inhibit mastication, phonation, the movements of the eyes, respiration; indeed, dyspnœa may become extreme and be associated with more or less violent asthmatic paroxysm. There is a marked leukocytosis (upward of 30,000) and a notable esosinophilia (often 50 per cent. of the leukocytes). (3) The period of regression, marked by exaggeration of all the symptoms; a second œdema, especially of the head ("big head"), may develop; anemia, emaciation, and a cachexia may supervene; or the parasites become encysted, the symptoms lessen, and the patient recovers.

Diagnosis.—The diagnosis may often be suspected from the fever, transient œdema of the eyelids, and sore muscles. These with marked eosinophilia make the evidence almost if not quite conclusive. In addition one should search for a source of infection, examine suspected pork for encysted larvæ; and examine the patient's stools and blood for the adult worms, and a piece of excised muscle (gastrocnemius or deltoid) for the encysted larvæ.

Prognosis.—The outlook depends upon the severity of the infection. In general it is good, better in children than in adults. The mortality does not average over 5 per cent., although in some local epidemics it may be very high.

Treatment.—The infection may be prevented by thorough inspection of pork at abattoirs, and by thoroughly curing and cooking all pork before it is eaten. If the infection is discovered or suspected early, the patient's gastro-intestinal tract should be thoroughly evacuated—by gastric lavage, and by calomel and saline cathartics. Perhaps thymol and beta-naphthol also might prove of service. Glycerin in large amounts has been recommended, in the hope that on account of its hygroscopic properties it may destroy the worms. Otherwise the treatment is symptomatic: hot applications or baths and morphine to relieve the pain, whiskey for its general supportive effect, etc. There is no known remedy that affects the parasite in the muscles.

Dracunculosis (Dracontiasis; Guineaworm Infection; Medina-worm Infection).—Infection with Dracunculus medinensis (Linnæus) is distinctly an Old World disorder, but occasional cases are observed in this country. The parasite is whitish or yellowish, 50 to 80 or more cm. in length, and 0.5 to 1.7 mm. in diameter; its anterior end is bluntly rounded, with a small terminal mouth and six papillæ. The uterus is enormously developed and filled with sharp-tailed embryos, 0.5 to 0.75 mm. long by 0.17 mm. in maximum diameter. Infection is probably acquired through the drinking water; possibly by way of the skin. After copulation the male probably dies, and the female wanders to the subcutaneous tissues. The *symptoms* consist of the production, by the gravid parasite, of a small, painful, blister-like swelling, particularly on the feet and ankles; this ruptures, leaving an ulcer, at the base of which the head of the parasite may be seen; chills, fever, nausea, and vomiting may occur. Healing does not ensue until the parasite has been expelled. The *treatment* consists of the injection into the ulcer of mercuric bichloride (1 to 1000) or excision of the parasite.

Filariasis.—Many filarial infections have been reported in man, but major interest centres in Filaria bancrofti (Filaria nocturna, Manson). The infection occurs in tropical and subtropical countries, but isolated cases have been observed in many regions of the temperate zone. The adult parasite, which occurs only in man, is a whitish or brownish, transversely striated worm, 44 to 95 mm. long by 0.1 to 0.26 mm. in diameter; the male has two spicules, 0.2 and 0.6 mm. long, an anogenital pore, 138 mm. from the tail; renal papillæ are uncertain, but apparently there are three pairs of postanal papillæ; the vulva of the female is 0.66 to 1.2 mm. from the head, the anus 225 μ from the tip of the tail; viviparous. The larvæ, which occur in a number of mosquitoes (Anopheles, Culex, Panoplites), are 300 to 340 μ long by 6.6 to 8.5 or 11 μ in diameter, and are provided with a sheath and sharply pointed tail. They are found in the circulating blood, and they show a more or less marked periodicity in that they are much more numerous during the night, but if sleep is reversed to the day-time, the periodicity also is reversed. Mosquitoes, while biting patients, swallow these larvæ, which then undergo development in the muscles, and finally, after fourteen to seventeen days, or by lower temperature, up to thirty-five to forty-one days from the time of infection, the worms reach a stage in which they are transmitted to man during the bite of the mosquito. Manson states that the organism may produce abscess, lymphangitis, varicose groin-glands, varicose axillary glands, lymph scrotum, cutaneous and deep lymphatic varix, orchitis, chyluria, elephantiasis of the leg, scrotum, vulva, arm, mamma, etc., chylous dropsy of the tunica vaginalis, chylous ascites, and chylous diarrhœa. The blood may reveal a lymphocytosis (up to 40 per cent.) and an eosinophilia (up to 18 per cent.). The *diagnosis* may be made by finding the larval filaria in the blood, urine, or chylous accumulations. Preventive measures comprise killing and avoiding mosquitoes. The older view that the infection was acquired

from the drinking water suggested its being boiled, but this view has been abandoned. As far as is known, medicinal *treatment* is without much avail, although good results are said to have attended the use of thymol, ichthyol, etc. The patient should receive supportive treatment, and it is perhaps wise to limit the fluid intake and give purgatives from time to time. Surgical intervention is indicated in some cases.

Filaria loa (diurna, Manson) is a small threadworm that circulates in the blood during the day, inhabits the connective tissues throughout the body, and is thought to be the cause of so-called Calabar swellings.

Filaria perstans, of which only the larvæ are known, is smaller than Filaria loa, and is said by Manson to be the cause of craw-craw, a papulopustular eruption of the West Coast of Africa; but like other filariæ it frequently is found in the blood in the absence of noteworthy symptoms.

Anguillula aceti (the vinegar-eel) and Leptodera pellio have been found in the human bladder and vagina; and Dicotophyme renale, the giant strongyle, or canine kidney-worm, has occasionally been found in man.

ACARIASIS.

(*Acarine Infections.*)

Ixodiasis Tick (Infection).—There are two families of ticks (Ixodoidæ)—Argasidæ, in which the head is subterminal in the adult; and Izodidæ, in which the head is terminal in the adult. The important species are Ornithodorus moubata, the agent active in transmitting Spirochæta duttoni, the cause of African tick fever (relapsing fever); Dermacentor venustus, Banks (andersoni, Stiles), and Dermacentor modestus (Banks), the agent active in transmitting the cause of Rocky Mountain spotted fever, or tick fever (which should not be confounded with the African tick fever); and Boophilus annulatus, the agent active in transmitting the cause of Texas fever. These and other ticks cling tenaciously to the skin, but on covering them with oil or vaseline (thus closing their breathing spaces) they release their hold and drop off.

Sorcoptic Acariasis (Sarcoptic Itch).—Infection with Sarcoptes scabiei is universal, involving man and animals. The parasite, as it occurs in man, is whitish yellow, round to oval, with transverse rows of small spines, and a number of longer bristles; the male measures 0.2 to 0.3 by 0.145 to 0.190 mm., and the female, 0.33 to 0.45 by 0.25 to 0.35 mm.; they bore irregular galleries, 0.5 mm. to 4 to 5 cm. or more in length, in the epidermis, especially on portions of the body where the skin is thin and soft, as on the flexor surface of the carpus, between the fingers, in the groins, at the knee, on the penis, breast, etc., and at points subject to pressure, as at the waistband. The female is found at the blind end of the gallery; she deposits her eggs (15 to 50 in number) and feces as she progresses. The eggs measure 140 μ and hatch in four to eight days; the parasite becomes mature twenty-eight days after birth. The male dies shortly after copulation. Infection takes place directly

from person to person through prolonged contact, indirectly through bedclothes, towels, clothes, etc., or by direct contact in handling animals. The main *symptoms* consist of itching, increased by heat, exercise, and rest in bed; and a papular, vesicular, or pustular eruption, the result partly of scratching and partly of the mechanical and chemical irritation provoked by the parasite. The *diagnosis* is readily made by detecting the galleries with the female at the end. The *treatment* consists of thorough cleansing of the affected part with green soap and hot water; and thorough inunction with mercurial ointment, sulphur ointment, or a compound naphthol ointment (naphthol, 15; prepared chalk, 10; green soap, 50; lard, 100; Kaposi). After the ointment has been allowed to act for four or five hours, the patient should take a warm bath and put on clean non-infected clothes.

Other mites may cause various disorders, such as Demodex folliculorum, which, invading the hair follicles, may set up nodular formations; Leptus americanus (irritans, autumnalis), the harvest mite, known also as the red bug, the harvest bug, or the jigger, which, burrowing into the skin of workers in the field in summer, may set up considerable irritation, and Pediculoides ventricosus, the cause of grain or straw-bed itch.

PARASITIC INSECTS.

Phthiriasis (Pediculosis).—There are three varieties of pediculidæ—Pediculus humanus (capitis), the head louse; Pediculus corporis (vestimenti), the body louse; and Phthirius pubis, the pubic louse. They are found only in persons of uncleanly habits. They cause irritation and itching and eczematous and pustular eruptions. The *treatment* consists of cutting the hair and saturating the infected parts with kerosene, or with carbolic lotions (2 per cent.), which are more destructive to the eggs (nits). Mercurial ointment is serviceable in the event of pubic infection.

Fleas.—Sarcopsylla penetrans (the burrowing flea, jigger flea, chigger) may burrow into the skin of the feet, but also elsewhere, and set up considerable irritation, suppuration, and ulceration. It should be taken out with a sharp knife; chloroform or mercurial ointment may be applied. Of jumping fleas, there are two that may attack man—Pulex irritans, the common or house flea, and Ctenocephalus canis, the cat and dog flea. These may act as ectoparasites, biting man; as an intermediate host for certain tapeworms; or as disseminators of plague. The house should be rid of fleas by sulphur fumigation, sprinkling about pyrethrum powder, or washing the floors with benzine or hot soapsuds.

Bed-bugs.—Two types of bed-bugs infect dwellings—Cimex lectularius, the common bed-bug, and Conorhinus sanguisuga, the Mexican bed-bug. Stiles recommends fumigation with flowers of sulphur, 2 pounds to 1000 cubic feet of space, as the best method of ridding a house of these pests.

Myiasis.—Compsomyia macellaria may deposit its eggs in wounds. The larva, known as the screw-worm, issues within a few hours, and may burrow into the tissues. Intestinal infection also may occur. Many other dipterous insects, mosquitoes, flies, beetles, etc., are of much and increasing medical importance, not so much because of the disorders that they may engender per se, but on account of the divers sorts of infection that they transmit. They are of special importance in tropical and subtropical countries, where the control of many of the known and unknown or ill-understood infections unquestionably depends upon a study of the life habits and other biological characteristics of various sorts of insects that undoubtedly act as transmitting agents.

INFECTIONS OF UNKNOWN OR DOUBTFUL ETIOLOGY.

VARIOLA.

(*Smallpox; Varioloid.*)

Variola is an acute, specific, infectious, and highly contagious disease, characterized by high fever, usually of sudden onset and of characteristic course, and an eruption consisting successively of macule, papule, vesicle, pustule, and crust.

Etiology.—Variola occurs at all ages (in the case of pregnant women sometimes implicating the fœtus), and affects all races, and the sexes indiscriminately. It usually prevails epidemically, especially during the late fall, the winter, and the spring; but sporadic cases are often encountered. One attack usually confers immunity, but undoubted cases of second and even third attacks have been reported. Some few persons are apparently naturally immune.

The exciting cause of the disease has not been isolated with certainty —although a number of supposedly causative microörganisms have been described. Recently some interest has been shown in the supposed etiological significance of a protozoön, Cytoryctes variolæ, described originally by Guarnieri, later studied by Wasielewski, and more recently investigated by Councilman, Magrath, and Brinckerhoff—who, together with Calkins, describe what they believe to be its life history. The streptococcus may be isolated from most of the lesions (primary, or essential, and complicating), and seems to bear some relation to at least certain phases of the disease. The contagium is present in the blood of the patient, the emanations and exhalations, the secretions and excretions, and especially the dried crusts. The disease is highly contagious throughout its entire course (possibly even before the occurrence of the eruption). Practically all persons exposed are attacked, unless

they are immune naturally (very rare), by a previous attack, or by vaccination. Infection is transmitted by direct contact or proximity, by a third person, or by fomites, especially clothes and house furnishings, to which the contagium clings with extreme pertinacity, and for a long time. Recrudescence of the disease from year to year in places where it has been for a time endemic can almost always be attributed to such fomites, or to an ill-defined case. The question of the aërial convection of the infection is still debated, though certain facts speak in favor of it. An initial mild case, or even varioloid, may be the starting point of a virulent and widespread epidemic. Infection is acquired by inhalation.

Pathology.—According to Councilman, Magrath, and Brinckerhoff, the specific lesion of variola (which is limited to the skin and the mucous membrane of the soft palate, pharynx, and œsophagus) consists of a focal, vacuolar degeneration of the stratified epithelium accompanied by serous exudation and the formation of a reticulum—which when fully developed constitute the characteristic multilocular pock, or pustule. In the skin the lesions begin with degeneration of the cells of the lower layers of the epidermis, accompanied by exudation, at first serous, later more or less cellular, the products of which are contained within the spaces of a reticulum formed by the degenerated cells. The exudate increases in amount and the spaces of the reticulum enlarge until its fibers finally rupture, and the lesion becomes a filled-out pustule. This development may take place wholly within the epidermis, and the fluid contents of the pock be separated from the corium by comparatively intact cells; or the corium may form the bottom of the pustule, in which case there is usually necrosis of the papillary border. Œdema, cellular infiltration, and hemorrhage of the corium may occur. The subsidence and repair of the lesion are accomplished by the removal of the fluid portion of the exudate by absorption and drying, and by the regeneration of the epidermis, in the course of which the residual mass of degenerated epithelial cells, leukocytes, and debris, enclosed between two layers of horny epidermis, the old and the newly formed, is exfoliated. The lesions of the mucous membrane are proportionate in extent and severity to those of the skin, but on account of the absence of a horny layer of epithelium the degenerated epithelial cells are cast off, and the vesicle within the epidermis is rarely seen, the pustule never. According to Councilman, Magrath, and Brinckerhoff the specific parasite of variola is found chiefly in the cells of the rete mucosum—its younger or cytoplasmic form in the protoplasm of the epithelial cells of early lesions and of such of the older lesions as are extending; its intranuclear form, for the most part, in more advanced lesions. According to the same authors, a constant, well-marked, and well-nigh characteristic lesion consists of proliferation within the hematopoietic organs, giving rise in the spleen, the lymph nodes, and the bone marrow to the formation of mononuclear, basophilic cells (which enter the circulation in large numbers), and in the lymph nodes and the marrow to phagocytic endothelial cells. Focal and interstitial cellular infiltration with mononuclear basophilic cells occurs constantly in

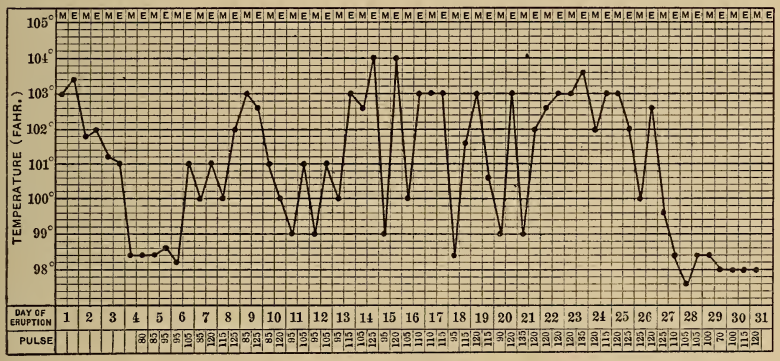
the testicle, and usually in the kidney, the liver, and the adrenal glands; in the testicle (in consequence of pressure and thrombosis) it causes focal nercosis—apparently specific to variola. Focal degeneration, toxic rather than anemic, and leading to necrosis, at times with hemorrhage, and accompanied by focal formation of phagocytic cells, is present in the blood-forming cells of the bone marrow, and constitutes a lesion almost pathognomonic. Diffuse toxic degeneration is present in the liver (where it is more marked than in any other infectious disease), the kidney, the adrenal gland, and the testicle. Toxic inhibition of cell differentiation is present in the bone marrow (absence of complete transformation of antecedent cells into polynuclear leukocytes) and in the testicle (absence of spermatogenesis). The paucity of polynuclear leukocytes in the specific lesions, in the focal degenerations, and in the bone marrow is so constant and so pronounced as to render it a striking peculiarity of the disease. The common secondary infections, due commonly to pyococci, do not differ pathologically from similar lesions occurring under different circumstances.

Symptoms.—The period of incubation is usually twelve days, though it varies from seven to fifteen days, and is commonly unattended by noteworthy prodromal symptoms (occasionally malaise). The onset is usually abrupt and quite characteristic, consisting of severe chills, high fever (103°, 104°, even 106° F.), severe lumbar pains, headache, and vomiting—which of themselves, in the presence of an epidemic, suggest the disease. Repeated chills or chilliness may occur within the first twenty-four hours. In children a convulsion not infrequently replaces the initial chill. In addition to the foregoing, in the early days of the disease, the patient is nervous and restless, his face is flushed, his eyes bright, and his skin hot and dry, though in some cases there is frequent and rather profuse perspiration; he complains of thirst, anorexia, and usually of constipation (though diarrhœa may be present in children); the pulse is rapid (100 to 140) and frequently dicrotic, and a systolic murmur, attributable to cardiac dilatation, sometimes develops; catarrhal inflammation of the conjunctivæ and of the respiratory tract (nose, throat, larynx, and bronchi) is rather common; and in many cases by the second or third day vertigo, stupor, or more or less delirium has developed. In severe cases coma and delirium may be initial symptoms. On the second day of the disease, in from one-sixth to one-fourth of the cases, so-called initial or prodromal rashes appear—measly, scarlatiniform, or petechial. Petechiæ may occur either alone or with the measly or the scarlatiniform rash. These, which often are quite indistinguishable from the ordinary rashes of measles and scarlatina, may be widespread, but they are usually localized to or involve especially the lower half of the abdomen, the outer aspect of the chest, the extensor and inner surfaces of the thighs and arms, and the natural folds of the body—the groins, axillas, elbows, etc. They usually fade at the end of twenty-four to thirty-six hours—except the petechiæ.

The period of invasion lasts three days, at the end of which time,

that is, on the evening of the third day or the morning of the fourth, the temperature declines, the lumbar pains and the headache abate, the general condition of the patient improves markedly, and the true variolous rash appears. In mild cases the temperature may fall rapidly to the normal; in severe cases this also may happen, but often the temperature, although it lessens with the outbreak of the rash, does not approach the normal until after the lapse of twenty-four to forty-eight hours (Fig. 11). The rash develops first on the forehead near or in the hairy scalp and on the wrists, and then gradually spreads over the entire body and sometimes to the mucous membranes in contact with the air (mouth, nose, conjunctiva, pharynx, larynx, Eustachian tube, trachea, œsophagus, anus, vagina, urethra). It is, however, always more marked on the face, scalp, wrists, hands, and fingers, and is less where the initial rashes were most marked. It consists at first of

FIG. 11


TEMPERATURE (FAHR.)
105°
104°
103°
102°
101°
100°
99°
98°
M E M E M E

the pocks become often extremely swollen and œdematous, so that the patient presents a most repulsive appearance; the cervical lymph nodes are usually much enlarged, and the spleen palpable; leukocytosis develops; albumin and casts are present in the urine in about two-thirds of the cases; and severe nervous symptoms, such as delirium and coma, not infrequently supervene. In favorable cases, desiccation begins on about the twelfth day, and by the fifteenth day is usually far advanced on the face. Gradually all the pocks dry up, and, as a rule, by the end of three weeks desquamation is complete. With the beginning of desiccation the secondary fever subsides, that is, it does not remain high for more than twenty-four to forty-eight hours; it gradually falls by lysis and reaches normal on or about the fifteenth day, and though irregular fluctuations may occur for some days, convalescence is now established. The amount and degree of pitting depend, as a rule, upon the severity of the infection, upon whether or not the skin papillæ have been destroyed. As a rule, the pits are pigmented for some time, and gradually whiten. The hair commonly falls out, but is soon restored.

In unfavorable cases, soon after pustulation occurs, a typhoid state with marked delirium usually develops; the pulse becomes rapid and feeble, coma, subsultus tendinum, etc., supervene, and with evidences of profound intoxication the patient dies.

The pocks on the mucous membranes, as a rule, do not proceed to suppuration, but rather to the formation of small ulcers. By their presence in the nose they occasion manifestations of coryza; in the mouth, fauces pharynx, and œsophagus, sore throat and dysphagia; in the Eustachian tube, purulent median otitis; in the larynx, œdema, perichondritis, and sometimes laryngeal obstruction; in the trachea and bronchi, tracheobronchitis; in the conjunctiva, conjunctivitis, sometimes progressing to keratitis with perforation, panophthalmitis, etc.

Varieties.—Several varieties of smallpox are described: (1) Discrete smallpox—in which the pocks are relatively few in number and discrete, and the general symptoms moderate in degree. (2) Confluent smallpox—in which the pocks are numerous and tend to coalesce, whence the greatest distortion of the countenance and limbs not infrequently occurs. Often the skin of the entire head and of the extremities becomes a large, superficial abscess. The general symptoms are severe—high fever, delirium, the typhoid state, etc.; death may ensue, or convalescence is prolonged. (3) Hemorrhagic smallpox: (*a*) in which the disease at the beginning pursues an ordinary course, but in which hemorrhages occur into the vesicles and pustules (variola hemorrhagica pustulosa). Death usually follows on from the seventh to the ninth day. Gradations, however, are observed between this type of the disease and the more mild cases, in which occasionally small hemorrhages occur into the pocks. (*b*) Petechial, black, or malignant smallpox (purpura variolosa), in which the blood dyscrasia is extreme, the initial rash markedly hyperemic (often purplish) and petechial; later the hemorrhages increase in size and involve the conjunctivæ and the mucous membranes (hematuria,

hematemesis, melena, menorrhagia, hemoptysis, etc.), the pulse becomes rapid, the respirations very much accelerated, and death often occurs as early as the third day—that is, before the true rash appears. (4) Varioloid, or modified smallpox, which occurs in persons that have been vaccinated. The initial manifestations are much like those of true smallpox (variola vera), though they are usually milder. The pocks are few in number, usually confined to the face and hands, and may not progress beyond the vesicular stage, though some of them may become pustular; there is little if any secondary fever, and rarely any scarring. (5) A verrucose form has been described—one in which the pocks dry up by the fifth or sixth day to a horny consistency—hornpox, wartpox, crystalline pox. (6) A variola sine eruptione has also been described; it is said to occur during epidemics, to affect young persons especially, and the initial manifestations (fever, headache, backache) only develop. It must be exceedingly rare, if it occurs at all. (7) Abortive cases, with severe initial symptoms, but subsequently a rapid favorable course.

Complications.—Complications, considering the common severity of the disease, are singularly few, though certain of the more common lesions may become aggravated and constitute real complications, such as laryngitis (which may lead to œdema and necrosis), conjunctivitis (and keratitis), and secondary infections, especially of the skin (furuncles, erysipelas, gangrene, etc.). Bronchopneumonia is common in the fatal cases. Pleuritis (often suppurative), otitis media, parotitis, orchitis, ovaritis, arthritis, and certain disorders of the nervous system attended by paralysis are sometimes observed.

Diagnosis.—The initial symptoms, abrupt onset, with severe chill, high fever, vomiting, severe lumbar pains, and headache, especially during an epidemic, suggest the disease. While the initial rashes may cause confusion in some cases, measles may be excluded by the absence of prodromes, of coryza, of conjunctivitis, of cough, and of Koplik's spots, and scarlatina may be excluded by the absence of sore throat, of unduly rapid pulse, of the hyperemic punctate rash in the fauces, and by the less uniform distribution of the cutaneous rash. Difficulty is often experienced in distinguishing between smallpox and chickenpox, but smallpox is suggested by a longer prodromal period, severe initial symptoms, long preëruptive stage, the abundance of pocks on the face, wrists, and hands (as contrasted with the trunk), by the hard and "shotty" character of the pocks which appear in one crop (within thirty-six hours), and are uniform in size, deep-seated, umbilicated, and multilocular, and do not show different stages side by side (as in varicella), by the occurrence of secondary fever and of leukocytosis, and by the absence of a recent successful vaccination. Typhus fever may be distinguished by the special etiological factors, by the macular and later petechial character of the rash, which is usually most abundant on the chest and the abdomen, and by the absence of remission of the fever upon the appearance of the rash. In pustular syphilis there is a history of syphilitic infection from six months to two years previously;

there are not the preëruptive manifestations of smallpox nor remission of the fever upon the appearance of the rash (though there may be some neuromuscular pains and slight fever); the cutaneous lesions are polymorphous; the pustular syphiloderm has usually been preceded by macular and papular syphiloderms; the pustules are usually discrete, come out in crops, are more abundant upon the trunk (relative freedom of the face), are rarely as deep-seated as in smallpox, though they usually have an indurated base; and there are frequently other evidences of syphilis. Impetigo contagiosa begins as vesiculo-pustules (not papules), and enlarging by peripheral extension, the lesions sometimes attain the dimensions of a ten-cent piece.

Prognosis.—The mortality varies much in different epidemics; it is commonly from 25 to 40 per cent. in unprotected individuals, but in recent years the disease seems to have become milder, or rather there have been many epidemics of mild smallpox. Severe, confluent, and hemorrhagic forms, as well as milder but complicated cases (especially in the debilitated), are likely to terminate fatally. The mortality of varioloid is from 1 to 3 per cent.

Treatment.—The patient must be isolated and the strictest measures of prophylaxis rigidly enforced (page 29). During the fever period the patient should be confined to bed, and should have an abundance of fresh air and a light nutritious diet—small amounts at frequent intervals. Attention to securing a sufficient diet is especially necessary during the later periods of the disease, when the abundant suppuration saps the vitality of the patient. Water should be given in large amounts—to relieve the thirst and to promote the elimination of the toxins by flushing the kidneys. The addition of the alkaline diuretics (potassium citrate, acetate, etc.) to the water subserves a useful purpose.

In the absence of a specific, treatment is largely symptomatic and supportive. Morphine is of much service in relieving the early pains, and later in controlling the cutaneous irritation and consequent nervous unrest, as well as, sometimes, the delirium. Vomiting, if continued, may be relieved by cocaine, cracked ice, champagne, or dilute hydrocyanic acid. The fever often calls for a diaphoretic febrifuge mixture (spirit of nitrous ether and solution of ammonium acetate), and cold sponging (occasionally the cold bath), which is of excellent service not only in controlling the fever, but in alleviating the evidences of toxemia (delirium, prostration, etc.). In some cases continuous warm baths (hours or days) are valuable. The throat pain may be relieved by cracked ice or orthoform lozenges (1 grain, 0.06 gram each). Stimulants (whiskey, brandy, strychnine, etc.) are often required in the later stages of the disease.

But little can be done to prevent pitting; puncturing the pocks, and the use of silver nitrate, carbolic acid, iodine, etc., for this purpose are almost if not quite useless. The use of hot or cold compresses, or of carbolized vaseline or oil is often grateful to the patient. Welch and Schamberg recommend painting the face once or twice daily with tincture of iodine. Crusts, when they form, should be kept moist by vaseline or

oil, which not only relieves the cutaneous irritation, but also prevents the dispersion of the infectious flakes. During the whole course of the disease the eyes and the mouth should be kept clean by the use of mild antiseptic lotions. The patient may be permitted to get out of bed as soon as the temperature is normal. Repeated bathing and the use of antiseptic soaps should be encouraged during convalescence. If pocks have occurred in the scalp, the hair should be cut very close.

Œdema of the glottis may necessitate tracheotomy. The severer forms of smallpox require considerable stimulation, but it is doubtful whether ergot, iron, gallic acid, etc., that have been administered in the hemorrhagic forms have been of any real service. Most of the special forms of treatment advocated from time to time, including the red-light treatment, have not proved of service in the hands of impartial observers of large experience.

VACCINIA.

(*Cowpox; Vaccination.*)

Vaccinia is an acute, specific, infectious, and highly contagious disease of bovine animals which, when inoculated into man (vaccination), produces fever, constitutional disturbance, and a specific eruption, consisting of papule, vesicle, pustule, and crust, and affords protection against smallpox.

Etiology.—Since Jenner introduced vaccination, in 1796, there has been discussion as to its nature—whether cowpox is an independent disease or smallpox modified by transmission through the cow. The weight of evidence, however, tends to confirm Jenner's opinion that cowpox is modified smallpox, that, at least, both are descended from a common ancestor, since (1) smallpox and cowpox afford immunity, the one to the other, and (2) human smallpox virus when inoculated into cows has produced cowpox (though cows are often refractory), and the lymph from such cowpox vesicles when inoculated into man has produced vaccinia, and occasionally even more or less generalized eruptions—which may be regarded as smallpox. Some support to this view is found also in the results (still awaiting confirmation) of the investigations by Councilman, Magrath, and Brinckerhoff regarding the supposed parasites of smallpox and of vaccinia. The many examinations of vaccine lymph have not disclosed with certainty the specific vaccine organism; divers cocci (including contaminating pyococci); bacilli (including occasionally contaminating tetanus bacilli), and certain supposed animal parasites have been detected by different observers. Whatever its exact nature, the virus is filtrable, and doubtless belongs to the class of ultramicroscopic organisms.

Method of Vaccination.—Cow lymph alone should be used, and only that obtained from trustworthy sources. The use of human virus has deservedly fallen into disrepute. During recent years the use of glycerinated cow lymph has become widespread, but many competent observers

still prefer the dried "points." Assuredly the lymph should be as free as possible from contaminating microörganisms—which has not always been the case.

The arm (near the insertion of the deltoid muscle) or the calf of the leg (frequently in girls) is usually selected for vaccination. The skin having been cleansed with water and soap (no antiseptics, since they destroy the vaccine), an area one-eighth to one-quarter inch in diameter should be scarified with a sterile scalpel or needle so as to remove only the epidermis, whereupon the vaccine should be rubbed in. One should be careful not to draw blood. A single area for inoculation is sufficient, though many European physicians prefer several, as many as five. After the vaccine has dried it should be covered with an aseptic dressing, and the wound thereafter treated aseptically. Most of the cases of serious inflammation of the arm following vaccination are attributable to secondary infection of the wound.

Symptoms.—The period of incubation of vaccinia is usually about forty-eight hours, and is commonly unattended by symptoms. On the third day a papule attended by swelling, redness, induration, and a feeling of tension becomes apparent at the site of inoculation; by the fifth or sixth day this has developed into an umbilicated, multilocular vesicle, the contents of which are at first clear but soon become turbid. Gradually the vesicle increases in size, and by the eighth day has reaced its maximum—when it is very itchy and surrounded by a wide zone of inflammatory induration and œdema. By the eleventh or twelfth day the contents of the pock are distinctly purulent, and the inflammatory zone may be quite extensive; the adjacent lymphatics are swollen and tender and inhibit the motion of the limb; and rarely almost if not quite the entire limb is swollen, but this is a manifestation of secondary infection. Soon after the twelfth day the inflammatory phenomena begin to subside, and by the fifteenth day a dried crust has formed, which usually drops off during the early part of the fourth week, leaving a pitted scar.

While the local lesions are at their height there is usually some elevation of the bodily temperature (100° to 102° F.), more or less malaise, slight headache, and occasionally chilliness.

Sometimes the evolution of the pock is unusually delayed. In other cases what may be mistaken for the true pock develops very early, but it does not present the characteristic appearances (vesiculation and umbilication); it soon drys, and by the seventh or eighth day the crust drops off. These so-called spurious vaccinations afford no protection against smallpox. In revaccinations the appearances of the pock are not infrequently atypical.

Complications.—Acland has arranged the possible complications as follows: During the first three days—erythema, urticaria, vesicular and bullous eruptions, invaccinated erysipelas; after the third day and until the pock reaches maturity—urticaria, lichen urticatus, erythema multiforme, accidental erysipelas; about the end of the first week—generalized vaccinia, impetigo, vaccinal ulceration, glandular abscess, septic

infections, gangrene; after the involution of the pocks—invaccinated diseases—syphilis. Complications, as a rule, are uncommon, the most common being septic infection attributable to lack of attention to the wound. Syphilis has rarely been transmitted, and, of course, never when cow lymph has been used. Tetanus has occasionally been transmitted, and has proved fatal.

The time for vaccination is during the first or second year of life, or immediately after birth if an epidemic prevails. Revaccination should be done before or about puberty, and every five or seven years thereafter, or oftener if an epidemic prevails.

The value of vaccination cannot be questioned—since where vaccination and revaccination are most thoroughly carried out smallpox is least common or is never seen (as in the German army, whence it has been banished), and where vaccination is most lax smallpox finds its readiest victims and exerts its greatest ravages.

VARICELLA.

(*Chicken-pox.*)

Chicken-pox is an acute specific, infectious, and moderately contagious disease, characterized by mild fever and a papulo-vesicular eruption.

Etiology.—Chicken-pox is essentially a disease of childhood, occurring usually before the seventh year, and attacking the sexes indiscriminately. It is rather uncommon in very young infants, but is probably more common in adults than is usually supposed. The disease generally occurs epidemically, but sporadic cases are rather frequently encountered. One attack usually, but not always, confers immunity.

The exciting cause of the disease has not been isolated, although it is unquestionably present in the skin lesions. Infection is usually transmitted by direct contact or proximity, but may be transmitted by a third person or by fomites. Infection is believed to be acquired by inhalation, and is much favored by previous ill health and overcrowding, such as obtains in asylums and some schools, etc. There is no longer any question of the non-identity of smallpox and chicken-pox—an attack of one of these diseases does not confer immunity from the other.

Pathology.—The characteristic lesion is the papulo-vesicular eruption. The lesions of the internal organs are those common to most of mild infections and toxemias.

Symptoms.—The period of incubation is commonly fourteen days, although it is said to vary from ten to nineteen days. The onset is usually mild in character and is often not preceded by prodromes, although slight fever, malaise, and slight pain in the back and legs may be present for a day; occasionally there are chilly sensations, and rarely convulsions. Often the rash, which comes out within the first twenty-four hours, is the first evidence of ill health. The rash appears first on the trunk (chest and back), but soon also on the neck, face, and forehead (near

or in the hairy scalp), and is sometimes encountered on the oral, pharyngeal, and laryngeal mucous membrane—even in some cases on the conjunctivæ and the genital mucosæ. The rash consists of small, reddish papules which speedily (within several hours) develop into vesicles; in some cases the papular stage may be apparently absent. The pocks, as a rule, are roundish or ovoid in outline, vary somewhat in size, contain clear fluid (though it may be turbid), are rather superficial, sometimes (but not often) umbilicated, although as desiccation progresses they become flattened and even somewhat depressed in the centre; though sometimes situated upon a slightly hyperemic base, this is often wanting, the pocks resembling clear blisters such as might be caused by drops of boiling water, and a peripheral zone of infiltration is not encountered. During the course of the second day the contents of the pock often become seropurulent, especially in ill-nourished children and in consequence of secondary infection. The individual pocks usually reach maturity at about the end of two days, and at the end of the third or fourth day desiccation begins; a scab soon forms, and is shed, as a rule, within two weeks, without leaving a scar. The rash is usually discrete, rarely confluent, and in consequence of its coming out in crops from the first to the third day, all stages in its development may be seen side by side. Usually not more than a dozen pocks, there may rarely be several hundred. In a few cases a scarlatinous rash precedes the development of the pocks. The temperature which is highest on the second and third days (as the rash comes out), rarely exceeds 102° F., though in severe infections in debilitated children it may reach 104° F. It falls by lysis as the rash fades. Some glandular enlargement, especially of the neck, may be encountered.

In unusual cases, the vesicles may become very large, forming bullæ and resembling pemphigus (varicella bullosa); in other cases hemorrhages into the cutaneous lesions and the mucous membranes occur (varicella hemorrhagica); and occasionally necrosis and gangrene about the pocks develop (varicella gangrenosa).

Complications.—Nephritis and adenitis sometimes occur. Secondary infection of the pocks sometimes leads to suppuration and subsequent scar formation, and occasionally to rather extensive streptococcic infection (erysipelas). Varicella is often associated with other infections (epidemically as well as individually), especially measles and scarlatina.

Diagnosis.—Difficulty is sometimes experienced in distinguishing between varicella and mild smallpox, but varicella is suggested by a short prodromal period, the abundance of pocks on the trunk (as contrasted with the face), the occurrence side by side of pocks in different stages of development (papule, vesicle, and crust), the apparent superficiality, bleb-like and unilocular character of the pocks, and a previous and rather recent successful vaccination. Impetigo, pustular eczema, etc., sometimes must be considered in the differential diagnosis.

Treatment.—The treatment consists of isolation (for two or three weeks) and general hygienic management (page 29). Carbolized vaseline or hot

boric acid solution should be applied to the pocks if there is much irritation. Medicines are rarely required, but stimulants should be administered to the weakly, and a generous diet is always advisable, especially during convalescence.

TYPHUS FEVER.

(Exanthematic Typhus Fever; Spotted Fever; Jail Fever; Ship Fever; Camp Fever; Famine Fever.)

Typhus fever is an acute, specific, infectious, and highly contagious disease, characterized by sudden onset, macular eruption, grave nervous symptoms, and a characteristic fever curve ending by crisis on or about the fourteenth day.

Etiology.—Typhus fever seems especially prone to attack those of adult life, but age, sex, race, season, and climate have little if any etiological significance. Formerly a widespread disorder and frequently occurring epidemically in different parts of the world, the disease is now seldom encountered, although sporadic cases occur in large cities from time to time. One attack usually confers immunity.

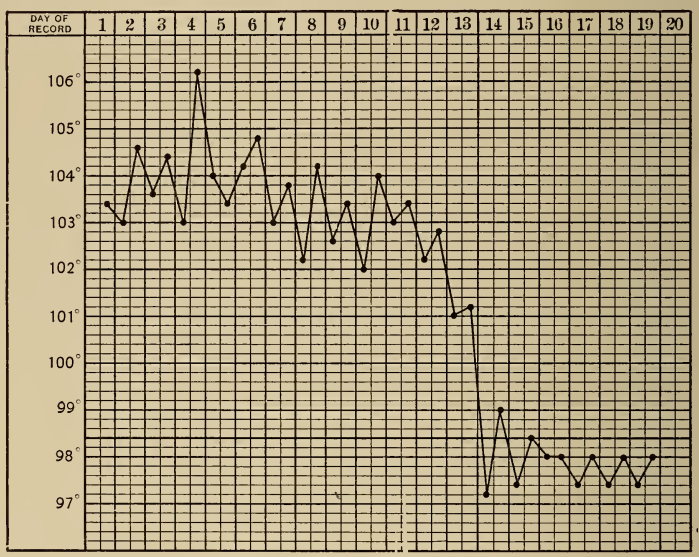
The exciting cause of the disease has not been isolated, although it it is probably present in the exhalations and evacuations of the patient, in the blood, the skin lesions, and the scales. Recently Krompecher, Goldzieher, and Argyan have described what they believe to be protozoa in the blood. The disease is extremely contagious, the contagium being transmitted directly, by a third person, and especially by fomites, such as clothes, bed linen, dishes, toys, etc., to which the contagium clings with extreme pertinacity. Anderson and Goldberger, and Ricketts and Wilder studying tabardillo, the typhus fever of Mexico, have transmitted the disease to monkeys; they have found that the virus is in the blood of the patient during the febrile stage, and that it is not filtrable; that monkeys that recover show an immunity comparable with that in man; and that the body louse (Pediculus vestimenti) may transmit the infection. The infection probably may also be acquired by inhalation, since physicians, nurses, and attendants upon the sick during epidemics are very likely to contract the disease. The likelihood of infection is increased by general insanitary practices, destitution, and overcrowding such as obtain in tenements, asylums, jails, ships, camps, etc.

Pathology.—There are no characteristic anatomical lesions, the lesions present being those common to most severe toxemias. Thus, the spleen is moderately enlarged and soft; the liver, kidneys, heart, voluntary muscles, nerves, etc., reveal parenchymatous and granular degeneration and necrosis; and there are marked hemolysis, subcutaneous petechial hemorrhages, and bronchial catarrh.

Symptoms.—The period of incubation varies from seven to twenty-one days, being usually twelve days. In some cases there are prodromal symptoms lasting a day or two, and consisting of general malaise, loss

of appetite, headache, vertigo, and sometimes coryza. The onset of the disease is usually sudden, and manifested by chill, high fever, headache, pains in the back and limbs, prostration, and sometimes vomiting. The temperature may reach 104° to 105° F., by the end of twenty-four or thirty-six hours, and it continues high with slight morning remissions (Fig. 12). The chill may recur for a day or two. With continuance

Fig. 12



The temperature in typhus fever, showing the decline by crisis. (Doty.)

of the high fever, marked nervous manifestations referable to toxemia soon appear, such as severe headache, vertigo, tinnitus aurium, spots before the eyes, etc. The patient becomes dull and listless, sometimes wildly maniacal. There is much thirst, the bowels are confined, and the urine is concentrated and may contain a small amount of albumin.

On the fourth or fifth day, by which time the temperature has reached its maximum, often 106° F., the characteristic eruption makes its appearance, first on the anterior fold of the axillæ, and the wrists, or the backs of the hands, then on the sides of the abdomen and chest, and finally on the trunk, including the back and the extremities, but being rare on the face. It is made up of two elements—rose-colored or dusky macules superimposed upon a subcuticular mottling, whence it much resembles measles, and has been spoken of as the mulberry rash of typhus fever. It comes out rapidly, within twenty-four hours usually, and not in crops as does the rash of typhoid fever. At first the macules, which fade almost imperceptibly into the surrounding mottling, disappear upon pressure, but they soon become darker and less deleble, and later (eight to tenth day) become hemorrhagic (petechiæ). The rash is rarely missed,

except perhaps in the mildest cases, in which, however, it is largely petechial.

During the course of the second week, concurrent with the change in character of the rash, profound toxemia (the typhoid state) develops. Delirium, often noisy at first, but later usually milder, muttering, and attended by sleeplessness, stupor (coma vigil), subsultus tendinum, and carphologia, continue. Prostration is extreme, and face expressionless and flushed, the pupils contracted, and the eyes injected. The temperature, though somewhat lower than during the first week, continues high (102.5° to 103.5° F.), the pulse is weak (120 to 130 per minute), small, and often irregular and dicrotic. The apex beat and the heart sounds are weak, and a soft, systolic murmur is common. There may be a slight leukocytosis. The tongue becomes dry and fissured, and later sordes collect on the lips, gums, teeth, and tongue. The breath and the body generally are malodorous (by some said to be characteristic). Bronchitis continues, and with failure of the heart, hypostatic congestion of the lungs may develop. The urine is usually concentrated and contains a little albumin, lessened chlorides, and is frequently voided involuntarily, although it may be retained. The spleen is usually enlarged and palpable. Bed sores not infrequently develop. In unfavorable cases, death ensues from exhaustion due to toxemia or from complications, the temperature often rising just before death to 108° to 109° F.

In favorable cases at the end of the second week, usually on the fourteenth day, and sometimes preceded by a precritical rise, the temperature falls by crisis, within twelve to twenty-four hours, to normal or below, the patient sweats profusely, toxemia lessens, the general symptoms improve, and convalescence ensues.

Varieties.—Mild and severe are sufficiently descriptive of the majority of cases. In certain abortive cases, the crisis occurs early and the eruption and delirium are not marked. In certain malignant or fulminating cases (typhus siderans) death ensues early under manifestations of profound toxemia, marked delirium, high fever, cardiac collapse, and degeneration of the viscera. In some cases the disease is protracted, the fever and other manifestations extending into the third week. An afebrile typhus fever has been described. Relapse is very rare.

Complications and Sequels.—Bronchitis, hypostatic congestion of the lungs, and bronchopneumonia are common. Gangrene of the lung sometimes develops. Thrombosis and embolism with consequent infarction of the viscera and gangrene of the extremities occur. Extensive gangrenous bed sores, suppuration of the glands and of other tissues, due to secondary infections, furunculosis, parotitis, meningitis, hemolysis, also have been observed. Hemiplegia or monoplegia, the result of thrombosis or embolism, and paralysis due to neuritis, sometimes follow. Rarely various forms of insanity persist for some time.

Diagnosis.—In cases of moderate severity, the diagnosis is apparent from the presence of an epidemic, exposure to infection, characteristic onset, high fever, and characteristic rash. The recognition of mild

cases, in the absence of an epidemic, requires a knowledge of the entire clinical course. Typhus, as contrasted with typhoid fever, is distinguished by a sudden onset with chill, high initial and more continued temperature, early prostration, severe nervous symptoms (delirium, stupor), a characteristic petechial rash (with diffuse mottling), more likely injection of the sclerotics, and the termination by crisis on or about the fourteenth day, and by the absence of abdominal symptoms, the presence of the Gruber-Widal reaction, typhoid bacilli in the blood, feces, urine, and rose spots. Cerebrospinal meningitis, measles, smallpox, uremia, purpura, acute infective endocarditis, and malaria have more or less resemblance in certain minor or major points to typhus fever, but regard for the clinical manifestations of each disease should soon settle the diagnosis.

Prognosis.—The mortality rate in epidemics varies from 10 to 20 per cent. The extent of the rash is said to bear a direct relation to the severity of the infection. Unfavorable signs are hyperpyrexia, extreme toxemia, and the different complications. The young usually recover.

Treatment.—The treatment in general does not differ from that of typhoid fever. The strictest isolation, thorough disinfection, and free ventilation (treatment in the open air), are imperative (page 29). The general management comprises the use of hydrotherapy and stimulants—hydrotherapy by sponging or bathing, large amounts of water to the patient to drink (much overlooked because of the stupor), and enteroclysis and hypodermoclysis to promote the elimination of toxins. Stimulants comprise the sponging and bathing just mentioned, alcohol (especially in subjects over twenty years of age), and strychnine, ammonium carbonate, camphor, etc. Coal tar derivatives should be avoided on account of the cardiac weakness. The food must be nutritious, easily digestible, given at stated intervals, bearing in mind that if we can support the patient for a stated time he is likely to recover from the self-limited disease. The complications are to be treated on general principles.

SCARLET FEVER.

(*Scarlatina; Scarlet Rash.*)

Scarlet fever is an acute, specific, infectious, and highly contagious disease, characterized by high fever, rapid pulse, sore throat, an erythematous rash, and a marked tendency to nephritis.

Etiology.—Scarlet fever is essentially a disease of childhood, attacking the sexes equally, and being especially prevalent before the tenth year (90 per cent. of all cases). It is rare in young infants, but more common in non-immune adults. The disease is endemic in most large cities and frequenty prevails epidemically, especially during the fall and winter. One attack usually confers immunity, although recurrences are observed. Some persons are naturally immune to the disease.

The exciting cause of the disease has not been isolated with certainty. Many observers view the disease as a modified streptococcic infection; the streptococcus may be isolated from most of the lesions, and assuredly plays an important part in the affection. Mallory has described a protozoan-like organism between the epithelial cells of the epidermis, and Duval has found the organism in blister-serum; but the proof is not yet conclusive that this is the specific infectious agent. The blood, nasopharyngeal and oral discharges, epidermic scales, and urine are infectious. The disease is highly contagious and spreads with rapidity, though not as rapidly or widely as measles. Infection in the great majority of cases is transmitted by direct contact or proximity to a patient, or a "carrier;" or by means of infected food, such as milk (responsible for a number of epidemics); occasionally by a third person, or by fomites. Infection is usually acquired through inhalation, sometimes through swallowing, and wounds. Patients with surgical wounds and puerperal women appear to be very susceptible to the infection, though most of the cases of supposed scarlatina described in such subjects are instances of streptococcic or staphylococcic infection.

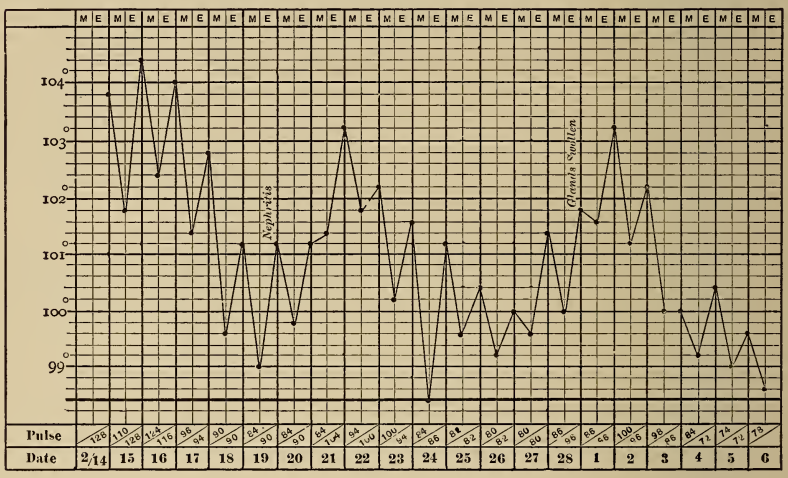
Pathology.—Scarlatina is devoid of characteristic lesions. The lesions in the nasopharynx are those of inflammation and ulceration; those in the skin, of hyperemia, which disappears after death. The lesions in the internal organs are those common to most toxemias, with sometimes special characteristics due to the frequent association of the streptococcus. The kidneys frequently show a rather characteristic lesion—a glomerulonephritis, though diffuse and hemorrhagic nephritis also occurs. Catarrh of the gastro-intestinal mucous membrane is common; the lymph nodes, especially those of the neck, not infrequently suppurate; and fibrinopurulent inflammation of the serous membranes is often encountered in fatal cases.

Symptoms.—The period of incubation is from one to seven days, usually from two to four days. The invasion is sudden—prodromal symptoms being entirely in abeyance or consisting merely of slight indisposition. The initial symptoms, which are quite characteristic and of themselves suggest the disease, consist of nausea, vomiting, high fever, 104° to 105° F. (Fig. 13), sore throat with pain on swallowing, swelling and tenderness of the glands at the angles of the jaw, and an unusually rapid pulse (140 to 180 per minute). Occasionally there is an initial chill; more often chilliness. Convulsions are common in young children. The throat is found to be acutely inflamed—the fauces, soft palate, and pharynx revealing a diffuse hyperemic redness and a fine, punctiform rash (even before the skin rash appears).

On the second day, often even within the first twenty-four hours, the characteristic rash appears—first on the neck and chest and then (within twenty-four hours) rapidly spreading over the entire body. This consists of a multitude of minute red points, for the most part corresponding with the points of emergence of the hairs, set in the diffusely hyperemic and slightly swollen skin. The hyperemic areas are usually confluent

and impart to the patient's skin an appearance suggesting a boiled lobster. The eruption is distinctly punctate in character—a character the more apparent the less the diffuse hyperemia; hence during the later stages of the disease. It disappears temporarily on pressure, and is usually most marked at the natural folds of the body—the groins, the bends of the elbow, the axillæ, and often on the lower part of the abdomen and on the inner aspects of the thighs. As contrasted with measles the face is often relatively unaffected.

Fig. 13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Nephritis
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2/14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 1 2 3 4 5 6

The temperature in scarlet fever, showing the decline about the fourth or fifth day, and a subsequent rise and irregularity, due to nephritis and swollen glands. (Musser.)

On the third day of the eruption, that is, at the end of about forty-eight hours, the disease is usually at its height. The fever remains high (104° to 105° F.) with slight morning remissions, the pulse frequent (140 to 160 per minute), and the entire integument presents a diffuse, bright scarlet hue (that serves to distinguish scarlatina from other exanthemas). The concomitants of fever and the expressions of toxemia are usually well marked: the patient is delirious at night and at least stuporous during the day, and the urine is reduced in amount, concentrated, of high specific gravity, and may contain a little albumin. The tongue, at first red at the tip and edges and covered on the dorsum with a grayish-white fur, through which the swollen papillæ project (strawberry tongue), soon loses the fur and frequently also the superficial epithelium, and presents a swollen, red, and irregular surface (raspberry tongue). The fauces exhibit different grades of inflammation: in the one case slight hyperemia with a punctate rash; in another case, a more severe inflammation especially of the tonsils, possibly a follicular tonsillitis; and in another case, pseudomembranous inflamma-

tion with more or less extensive induration and suppuration of the adjacent tissues. The glands at the angle of the jaw, swollen in all cases, frequently become exquisitely tender and may suppurate. The appetite is lost. The spleen may be palpable. Leukocytosis is common and may be marked.

Having lasted four or five days (in mild cases, two or three days), the rash begins to fade, the throat manifestations abate, and the fever falls by lysis—reaching the normal, in moderately severe cases, by the eighth or ninth day—in mild cases earlier, in severe cases and in the event of complications, later. The subsidence of the rash is followed by a branny or flaky desquamation, which begins (as did the rash) on the neck and chest, and is usually proportionate to the severity of the rash. In severe cases veritable casts of the hands and feet may be shed; sometimes even the hair and nails are lost. Desquamation is usually complete at the end of fifteen or twenty-days, but may be prolonged until the seventh or eighth week.

Varieties.—Depending upon the severity of the infection, the following varieties of scarlatina may be recognized:

1. Mild scarlatina, in which the general and local symptoms are mild, and in which the rash is slight and lasts but a day or two. In some cases it may be absent—scarlatina sine eruptione. In some cases the nature of a mild sore throat is unsuspected until the skin desquamates, or until nephritis develops, or until an epidemic attributable to it ensues.

2. Severe scarlatina, in which the general and local symptoms are severe: sudden onset, high fever (108° F.), scanty albuminous urine, marked prostration, severe nervous manifestations, headache, rapid feeble pulse, heart weakness, delirium, convulsions, coma, etc.—the so-called atatic form. This form, the expression of severe toxemia, is frequently fatal within a day or two—in some cases even before the appearance of the rash (during the period of invasion).

3. Malignant scarlatina, of which we may distinguish: (*a*) The hemorrhagic form—in which hemorrhages occur into the mucous membranes and the skin. Often the hemorrhages are petechial, but frequently ecchymoses, involving large areas, develop; and epistaxis, hemoptysis, hematuria, etc., may occur. Intoxication is severe and death usually results on the second or third day. (*b*) The anginose form—in which the throat symptoms assume the predominance. Ulcerative and pseudomembranous inflammation of the tonsils and fauces, with marked fetor, induration and suppuration of the adjacent cellular and lymphatic tissues ensue. The lesions may spread to the nose and the larynx, leading to death from asphyxia; or the cellular inflammation may become widespread, and lead to Ludwig's angina, or perforation of the carotid artery, etc. Otitis media is common. Bacteremia is the rule, and death speedily follows in most cases. The throat lesions, frequently diphtheroid, may sometimes reveal the Klebs-Loeffler bacillus.

The rash sometimes exhibits peculiarities. Instead of becoming diffuse, the hyperemic areas may remain discrete—scarlatina maculosa;

sometimes papular elevations occur—scarlatina papulosa; sometimes sudaminal vesicles appear—scarlatina miliaris; the vesicles may become purulent—scarlatina pustulosa; or large blebs may form—scarlatina pemphigoidea.

Complications and Sequels.—The most important complications and sequels are nephritis and inflammation of the serous membranes. In addition to the albumin present in most cases and attributable to the toxemia, a true nephritis may occur. It may develop during the course of severe cases, but is quite common after mild cases, especially during convalescence (third or fourth week)—postscarlatinal nephritis. The onset is usually manifested by dropsy, but sometimes by lessening in the amount of urine or by uremia. The œdema often goes on to anasarca and effusions within the serous cavities and the meninges. These may lead to death, but usually recovery ensues after several weeks, although convalescence may be protracted for several months. An œdema without nephritis, and attributed to impoverishment of the blood, has been described. A septic nephritis, as part of a general pyemia, sometimes occurs. Chronic nephritis rarely develops out of the acute lesions, but the kidneys may remain susceptible for a long time, and in later life exposure to appropriate etiological factors may result in the ready production of a serious and even fatal nephritis.

Of the inflammations of the serous membranes, pleuritis and pericarditis are common, and usually fibrinopurulent in character. Simple and malignant endocarditis, as well as myocarditis, may occur. Meningitis may develop and remain for a time unsuspected, especially in the event of convulsions. Pains in the joints are not uncommon—scarlatinal synovitis (miscalled rheumatism). Usually, an expression of the toxemia, the pain subsides with the toxemia; occasionally it persists longer, and the lesions may go on to suppuration (usually mono-articular). An associated chorea has been observed. Suppurative otitis media, the result of extension of inflammation along the Eustachian tube is common, and answerable for much of the deafness of later life. If neglected it may lead immediately to mastoid disease, sinus thrombosis, cerebral abscess, etc. An otorrhœa or rhinorrhœa persisting after apparent convalescence, even if non-purulent ("chronic carriers"), is answerable for the propagation of the disease and even for some epidemics. Suppurative adenitis, especially of the neck, is rather common. Sudden death from acute cardiac failure sometimes occurs during convalescence.

Concurrent infections are not infrequent—especially diphtheria, chicken-pox, measles, pertussis, erysipelas, typhoid fever, etc.

Diagnosis.—The diagnosis is usually easy. The short period of incubation, the characteristic initial symptoms, especially the early sore throat, the vomiting, the rapid pulse, the punctate hyperemia of the mucous membrane of the mouth and fauces, followed by the characteristic skin eruption, serve to exclude other diseases. From measles scarlatina may be excluded by the sore throat (as contrasted with general catarrhal symptoms), shorter preëruptive period, the punctiform rash in the throat,

the absence of the Filatow-Koplik's spots, the more uniform erythematous rash (as contrasted with the blotchy or mottled rash of measles), the relative freedom of the face, the raspberry tongue, the undue acceleration of the pulse, and the presence of leukocytosis. In rötheln, the onset is less abrupt, there is rarely initial vomiting, the fever is less high, the pulse is less rapid, the rash is not punctiform, there is no raspberry tongue, the general symptoms are less marked, and there is little tendency for the development of nephritis. In scarlatina the throat lesions sometimes become "diphtheroid," and may not be distinguishable from those of true diphtheria, except by bacteriological examination. Diagnostic difficulties are sometimes enhanced by an erythematous rash that occasionally occurs in diphtheria. However, this is uncommon, and is darker and of shorter duration than the true scarlatinous rash, and is usually confined to the trunk. The two diseases not infrequently co-exist. In acute tonsillitis there is rarely an erythematous rash, but this is not punctiform, there is no raspberry tongue, no undue acceleration of the pulse, and no epidemic. Acute exfoliative dermatitis is often difficult of differentiation, but is suggested by absence of throat symptoms and of the characteristic tongue, and by the rather marked tendency to recurrence, the earlier commencement of desquamation (even before the eruption has faded), the frequent involvement of the hair and the nails, and the comparative rarity of involvement of the kidneys. Drug rashes sometimes follow the administration of belladonna, quinine, potassium iodide, antitoxic serums, etc., and ptomaine rashes may occur after the use of tainted oysters, fish, etc., but these rashes are usually slight and transient (though they may be diffuse and marked), they are not punctiform, and there are none of the characteristic symptoms of scarlatina.

Prognosis.—The mortality varies much—from about 5 per cent in the mildest epidemics to 30 per cent. in the more severe. The death rate is higher the younger the child, and in general the poorer the social conditions. Unfavorable symptoms in individual cases are high fever, marked toxemia (delirium, etc.), hemorrhages, severe throat lesions, laryngeal obstruction, and nephritis.

Treatment.—The patient must be isolated and the usual prophylactic measures enforced (page 29). Specific therapy is still a hope of the future. Some good results have followed the use of a polyvalent antistreptococcic serum. More recently streptococcic vaccines have been employed for prophylactic and curative purposes—with encouraging result. In the absence of specific treatment, the special function of the physician is to guide the child through the attack, and to prevent the development of complications. Mild cases, as a rule, pursue a favorable course under the beneficent influence of rest in bed, flannel garments (to lessen the likelihood of chill), an abundance of fresh air, a milk diet, considerable water (to promote the elimination of toxins), and regulation of the bowels. In all cases it is wise to insure a daily evacuation of the bowels, to administer a mild diuretic and diaphoretic febrifuge mixture (such,

for instance, as is given on page 250), to give the child a warm bath twice daily and to follow this by oil inunctions. In the event of high fever and delirium, an ice bag should be applied to the head, and the bathings should be more frequent (every three hours). The bathing is much preferable to the use of antipyretic drugs. An alcohol rub at night often promotes a restful sleep. Stimulants (brandy, whiskey, strychnine, ammonium preparations, etc.) are called for in the event of marked toxemia (prostration, marked delirium, subsultus tendinum, the typhoid state, coma, etc.). The bromides are often serviceable in the wakefulness that sometimes attends the nervous irritability. J. C. Wilson recommends the routine use of chloral, in dosage sufficient to produce light somnolence, believing that it promotes diuresis and lessens the likelihood of nephritis, favorably modifies the nervous manifestations, and allays the itching. Robert Milne recommends thorough rubbing of the body, from the crown of the head to the soles of the feet, morning and evening, for the first four days, and then once daily until the tenth day, with 10 per cent. eucalyptus oil; and swabbing the throat, every two hours, for the first twenty-four hours, with 10 per cent. carbolic-acid oil—stating that this treatment modifies the severity of the attack, prevents nasal, oral, and renal complications, and abolishes the infectivity of the disease.

The throat manifestations, as a rule, do not call for special treatment, but cleansing preparations should always be used—to reduce if possible the bacterial content of the throat, but more especially to remove all secretion that may tend to obstruct the inlet to the Eustachian tube and thus favor the development of middle-ear disease. Normal salt solution, Dobell's solution, hydrogen peroxide (15 per cent), potassium chlorate (2 per cent.), or potassium permanganate (2 per cent.), may be used for this purpose. Many physicians are partial to the use of iron which may be employed as follows: Tincture of iron chloride ($\frac{1}{2}$ ounce, 15 c.c.), glycerin ($\frac{1}{2}$ ounce, 15 c.c.), and water (1 ounce, 30 c.c.). 10 to 15 minims of carbolic acid may be added to the mixture. In severer cases Löffler's solution is serviceable: Menthol, 10 c.c.; toluol, 36 c.c.; tincture of iron chloride, 4 c.c.; and absolute alcohol, 60 c.c. This may be applied by means of a cotton swab. Should cultures from the throat reveal the diphtheria bacillus antidiphtheritic serum should be used without delay.

During desquamation the child should be bathed daily with warm water and an antiseptic soap and then annointed with vaseline or oil. Especial attention should be paid to the ears, and in the event of pain appropriate measures, including early puncture of the drum membrane, should be instituted so as to obviate, if possible, deafness in later life. Iron and the bitter tonics are useful during convalescence. The child should be confined to the bed for at least ten days after the temperature is normal. During the entire convalescence one should always be guided by the knowledge that nephritis may develop (after exposure) three or more weeks after the temperature has reached the normal, and that the

child is perhaps infectious as long as there is any desquamation and certainly as long as a naso-pharyngeal or aural discharge persists. The complications are to be treated on general principles.

MEASLES.

(*Morbilli; Rubeola.*)

Measles is an acute, specific, infectious, and highly contagious disease characterized by catarrhal inflammation of the conjunctivæ and the respiratory mucous membrane, fever, and a peculiar maculopapular rash.

Etiology.—Measles is essentially a disease of childhood, attacking the sexes equally, and being especially prevalent between the second and the eighth year. It is unusual under six months of age, but it is more common in non-immune adults than certain other infective diseases, such as scarlatina. Recurrences are not uncommon, but one attack usually confers immunity. In many large cities the disease is endemic, and it frequently prevails epidemically, especially during the fall and winter.

The exciting cause of the disease has not been isolated, although it seems to be present in the emanations from the patient, especially the breath, the nasopharyngeal secretions, and the desquamations. The disease is highly contagious and spreads with extreme rapidity. Infection may be transmitted directly, or by a third person, or by fomites. It is acquired through inhalation, and is much favored by overcrowding, such as obtains in close, unsanitary dwellings, school-rooms, etc.

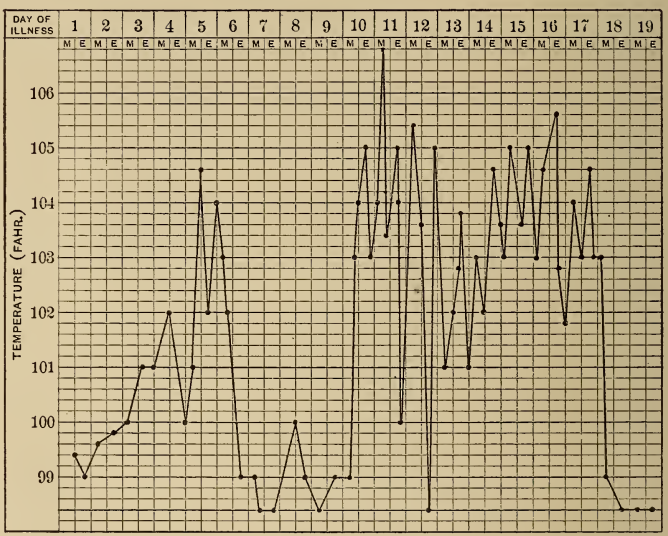
Pathology.—Measles is devoid of characteristic lesions. Catarrh of the conjunctivæ and the respiratory mucous membrane and hyperemia of the skin with slight if any cellular exudation occur. In severe and fatal cases bronchopneumonia, capillary bronchitis, atelectasis, pleuritis, and swelling of the bronchial lymph nodes and of the mucous membrane of the gastro-intestinal tract may be encountered.

Symptoms.—The period of incubation is from seven to fourteen or eighteen days, the average being about ten days. The invasion, usually, is rather abrupt, with chilliness (or convulsions or vomiting in infants) moderate fever, 101° to 102° F. (Fig. 14), well-marked coryza, and catarrhal inflammation of the conjunctivæ and the respiratory mucous membrane. The child appears to suffer from a cold in the head. It has a profuse nasal discharge, its eyes are injected, it complains of headache, photophobia, lacrymation, and sneezes frequently. Within twenty-four hours, as a result of extension of the catarrhal inflammation to the pharynx and bronchi, a cough develops. The fever and catarrhal manifestations continue and lead to anorexia, increase of the cough with bronchial secretion, wheezing, and dyspnœa. There is usually a slight remission of fever on the second and third days.

On the fourth day when the general symptoms are at their height, and the fever often 103° to 104° F., the characteristic eruption appears,

first on the forehead or cheeks, and spreads, within a very short time, over the entire body. The eruption consists of minute purplish-red papules, which are slightly elevated, and tend to coalesce to form crescentic figures with intervening areas of clear skin, giving rise to a blotched or mottled appearance. Usually confluent, the eruption may remain discrete, especially on the trunk and limbs. In severe cases it is quite dusky in appearance and the affected skin is much swollen. Being hyperemic, it usually disappears on pressure, but hemorrhages may occur. In most cases there is usually swelling of the lymph nodes, especially those of the neck.

Fig. 14


DAY OF ILLNESS
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19
M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E
TEMPERATURE (FAHR.)
106
105
104
103
102
101
100
99

The temperature curve in measles, complicated by pneumonia, in a boy, aged five years. (Tuley.)

Preceding the rash by twenty-four, sometimes forty-eight, hours or more, the Filatow-Koplik spots, a characteristic feature of the disease, may be observed on the buccal and the labial mucous membrane. These consist of small, irregular spots of bright red color, in the centre of which, in strong daylight, a minute bluish speck may be seen.

By the fifth day, in the majority of cases, the fever, the general symptoms, and the eruption have reached their height. By the sixth or seventh day, after having lasted two or three days, the rash begins to fade as it developed, from the face first, and it is succeeded by a branny desquamation. The nasal discharge lessens, the cough becomes looser, and there may be free expectoration. By the eighth day, usually, the temperature, having fallen by rapid lysis, is normal and convalescence is established.

Varieties.—In unusual or atypical cases, the rash may occur unusually early, within thirty-six hours, or it may be unduly delayed (sixth day). In other cases of house epidemics the child may manifest the initial coryza and no eruption at all may appear. Hemorrhagic measles is a severe infection, attended by petechial eruption, sometimes hemorrhages from the mucous membranes, and marked prostration from toxemia. It is often fatal, and occurs usually in institutions or barracks where the sanitary arrangements are bad.

Complications and Sequels.—Capillary bronchitis or bronchopneumonia, which is responsible for most of the fatalities, with atelectasis, may result from extension of the ordinary bronchitis. The less common complications are lobar pneumonia, pleuritis, severe catarrhal pseudomembranous inflammation of the mouth and larynx, œdema of the glottis, catarrhal inflammation and suppuration of the middle ear, gastroenteritis, gangrene of the mouth, vulva, etc. Tuberculosis of the lungs and of the bronchial and mediastinal lymph nodes is an important and not infrequent sequel. Arthritis and paralysis due to myelitis and multiple neuritis (polyneuritis) have been observed.

Diagnosis.—The diagnosis is usually easy. The presence of an epidemic, initial coryza, fever, and catarrh of the respiratory mucous membrane, suggest the disease, even before the appearance of the pathognomonic Filatow-Koplik spots and the characteristic eruption. From scarlatina measles may be distinguished by the longer preëruptive stage, the general catarrhal symptoms (instead of mere sore throat), the Filatow-Koplik spots, the blotched or mottled appearance of the rash with special involvement of the face (as distinguished from the more uniform erythema of scarlatina with relative freedom of the face), the absence of leukocytosis (except in the event of complications), the absence of undue acceleration of the pulse, absence of the characteristic scarlatinal tongue, and the fall of temperature after the rash is well out.

It may be difficult or impossible in some cases to differentiate measles from rötheln, but rötheln is suggested by less fever, shorter preëruptive stage (one or two days), less catarrhal symptoms, more sore throat, general uniform distribution of the rash, absence of crescentic arrangement, and very slight desquamation. The presence of fever and catarrhal symptoms serve to differentiate measles from urticaria and multiform erythema, as well as from different drug eruptions, in which the history also may be of service. The initial eruption of smallpox may be mistaken for measles, but the differential diagnosis does not long remain in abeyance. Four days of fever and catarrh usually exclude variola, varicella, rötheln, and scarlatina.

Prognosis.—The prognosis is favorable in simple cases. Complications, such as bronchopneumonia, hemorrhagic rash, etc., render the prognosis grave.

Treatment.—Hygienic regulations (page 29), rest in bed, and light nutritious diet are most important. Otherwise the treatment is purely symptomatic. The fever is rarely high enough to demand special

attention, but the general comfort of the child may be enhanced by daily tepid sponging or a tepid bath, especially if the temperature is about 103° F. Should the rash not appear, a hot bath and warm drinks will please the elderly nurse and do no harm: they are believed to hasten the development of rash. The distressing cough and the general condition of the child may be relieved by a mild, sedative and diaphoretic fever mixture, such as the following:

℞—Spirit of nitrous ether	3 drams	12	00
Camphorated tincture of opium	2 drams	8	00
Wine of ipecac	1 dram	4	00
Solution of ammonium acetate, sufficient to make . .	3 ounces	100	00—M.

S.—To a child, two years old, one teaspoonful (5 c.c.) every three hours.

During desquamation the skin should be anointed daily with vaseline, olive oil, or cacao butter, and the daily bath continued. Great care must be exercised during convalescence, especially if the child is weakly. Nutritious diet, milk, tonics, such as iron, quinine, and strychnine, hypophosphites, and cod-liver oil subserve a useful purpose.

RÖTHELN.

(*Rubella; German Measles; Rubella Notha; Epidemic Roseola.*)

Rötheln is an acute, specific, infectious, and highly contagious disease characterized by slight fever, coryza, and a maculopapular rash.

Etiology.—Rötheln occurs during childhood and in adult life, but it is more common during childhood, and attacks the sexes equally. Sporadic cases are common, but the disease often appears epidemically. One attack usually confers immunity, but recurrences have been observed.

The exciting cause of the disease has not been isolated, though it seems to be present in the emanations from the patient, especially the nasal and oral discharges and the skin lesions. The disease is highly contagious and spreads with extreme rapidity. Infection may be transmitted by direct contact, by a third person, and by fomites. Infection is acquired by inhalation. The patient is infectious from the period of incubation until convalescence.

Pathology.—There are no characteristic anatomical lesions.

Symptoms.—The period of incubation is usually from ten to twelve days, but it has been reported to vary between three days and three weeks. The invasion is usually mild, and may be devoid of prodromal symptoms, although chilliness, mild fever (rarely more than 100° F.), malaise, headache, backache, coryza, sore throat, and dry cough may be observed for a day or two. Usually on the second day, that is, at the end of the first twenty-four hours (sometimes earlier), the characteristic rash appears—first on the oral and faucial mucous membrane and the face, then on the chest, and within twenty-four hours spreads over the entire body. In some cases, however, it remains curiously restricted to certain

parts of the body, and may be observed only on the roof of the mouth and the fauces. The eruption consists of rosy-red, slightly elevated papules, that are usually discrete, though they may become confluent. They show little tendency to form crescentic patches, and the intervening skin is often hyperemic. With or just before the appearance of the rash the temperature may become a little more elevated, and it remains usually between 100° and 102° F. while the rash is out; the pulse is proportionately rapid; coryza, conjunctivitis, sore throat, and evidences of bronchitis become more marked; and the cervical lymph nodes (rarely other lymph nodes) are enlarged. The rash lasts usually two to five days, gradually fades, and is succeeded by a very slight branny desquamation and a brownish discoloration of the skin, which disappears after a day or two. With the fading of the rash the general symptoms lessen, and convalescence is usually established at the end of a week.

Complications.—Severe bronchitis and bronchopneumonia sometimes occur.

Diagnosis.—Considerable confusion in diagnosis often occurs, inasmuch as rötheln exhibits the sore throat of scarlatina and the rash of measles. From measles, however, rötheln may be distinguished by the less fever, shorter preëruptive stage, less catarrhal symptoms, more sore throat, general uniform distribution of the rash, absence of crescentic arrangement, and very slight desquamation. From scarlatina rötheln may be distinguished by the less abrupt onset, less high fever, less rapid pulse, absence of vomiting, absence of the strawberry tongue, absence of a punctiform rash, and the non-occurrence of nephritis. Dukes' so-called "*fourth disease*" is a disorder of which the incubation period is that of German measles; prodromes are usually absent; the onset is abrupt, with moderate fever, sore throat, and a rash that may cover the body within a few hours, although the face may remain free; the rash is brighter red than that of scarlatina, and may be followed by slight desquamation (more than in rötheln), rarely by marked desquamation; there are no sequels; and the disease does not protect against rötheln or scarlet fever. Whether or not it is an independent disease has not been determined. *Infectious erythema* is a disorder described by Escherich, and characterized by a rose-red, maculopapular (morbilliform) rash developing especially on the face and the flexor surfaces of the extremities; it occurs epidemically in children between the ages of four and twelve years; there may be no subjective symptoms; the rash usually fades in five to six or ten days, and is not followed by desquamation.

Prognosis.—Recovery is the rule.

Treatment.—The treatment is similar to that of measles.

GLANDULAR FEVER.

(*Drusenfieber, Pfeiffer.*)

Glandular fever is an acute, infectious, and moderately contagious disease, characterized by fever and swelling and tenderness of the lymph nodes.

Etiology.—Glandular fever is essentially a disease of childhood, though a few cases have been observed in adults. It often prevails epidemically, especially in the late fall, the winter, and the spring, but sporadic cases are sometimes observed. A specific exciting cause of the disease has not been isolated. Streptococci, staphylococci, influenza bacilli, and pneumococci have been found in the oral secretions in different cases, and suggest the opinion that the disease is probably not always due to the same cause. The disease is contagious, but how the contagium is transmitted has not been determined with certainty. Infection is believed to be acquired by inhalation, the causative bacteria acquiring a nidus in the mouth and the upper respiratory tract, whence they readily gain access to the regional lymph nodes.

Pathology.—The conspicuous feature of the disease is inflammatory swelling of the lymph nodes, especially the cervical, but sometimes also the axillary, inguinal, and mesenteric.

Symptoms.—The period of incubation is not definitely known, but it is probably several days. The onset is usually abrupt, with chill or chilliness, fever (101° to 103° F.), headache, restlessness, sometimes nausea and vomiting, and pain in the neck, especially on motion. Sometimes there is complaint of sore throat, and slight swelling and redness of the tonsils may be discovered. Usually on the second day the enlarged lymph nodes become apparent—swollen, painful, varying in size from that of a pea to a walnut, and not infrequently associated with periglandular œdema. Cough and oppression in the chest sometimes suggest involvement of the bronchial lymph nodes. The mesenteric nodes are sometimes palpable, and explain the rather common abdominal pain and tenderness. The bowels are usually constipated, and pains in the joints are not infrequent. The fever continues, as a rule, about a week and falls by lysis; the lymph-node enlargement lasts from two to three weeks, and gradually subsides. Suppuration is rare. Cutaneous erythemas, otitis media, bronchopneumonia, and nephritis are the more common complications, and are answerable for the few fatalities.

Diagnosis.—The diagnosis is usually easy, but occasionally, especially in the event of the cutaneous rash, one must be careful to exclude scarlatina.

Treatment.—Rest in bed during the period of fever at least, a liquid diet, an antiseptic mouth wash, a preliminary course of calomel, and a mild diuretic fever mixture (such as that mentioned on page 250) are all that are required—since we are unable directly to influence the duration of the disease; but one should endeavor to prevent complications.

RHEUMATIC FEVER.

(*Acute and Subacute Rheumatism; Articular Rheumatism.*)

Rheumatic fever is an acute and subacute (possibly specific) infectious and non-contagious disease, characterized by fever, multiple arthritis, sweating, and a tendency to inflammation of the serous membranes and the fibrous tissues of the body.

Etiology.—Rheumatic fever occurs especially in adults between the ages of fifteen and thirty-five years, but no age is exempt, and the disease has been observed in very young children. In children, however, the arthritic manifestations are often quite subsidiary to the cardiac. The disease is especially common in those exposed to the influences of wet, cold, and sudden changes in temperature—hence males rather than females, and among men, laborers, coachmen, drivers, sailors, etc., and among women, serving-maids, washerwomen, etc. Between the ages of ten and fifteen years, however, girls are more commonly affected, while prior to the tenth year the sexes suffer equally. The disease is endemic in temperate climates, and prevails especially during February, March, and April, but also during the colder months. Occasionally it exhibits epidemic characteristics. One attack, rather than conferring immunity, predisposes to subsequent attacks.

The exciting cause of the disease has not been isolated, although etiological importance has been attributed to a number of different microörganisms, and the anatomical and clinical characteristics of the disease permit of no doubt of its being an infection. Possibly the same etiological factor is not operative in all cases, and we may yet be able to differentiate a true rheumatic fever from other pseudorheumatic, pyemic, or toxic disorders with arthritic manifestations. Although the disorder has been attributed to streptococci, staphylococci, etc., the most likely causative factor is a diplococcus studied by Poynton and Paine, who isolated it from the joint lesions, the rheumatic nodules, the blood, etc., and were able to produce arthritis, valvulitis, etc., in rabbits. How infection is acquired has not been definitely determined for all cases, but there is an increasing disposition to regard the tonsils as the portals of entry for the infection in many, if not all, cases: the clinical relation is undoubted.

Pathology.—The inflammatory manifestations in the joints, serous membranes, and fibrous tissues exhibit scarcely anything characteristic, with the possible exception of the rheumatic nodule (found in tendon sheaths and over bony prominences), which consists of a central area of necrosis, surrounded by swollen and œdematous connective tissue and cellular exudation. The articular fluid is turbid, albuminous, and usually contains a few fibrin flakes and leukocytes.

Symptoms.—The period of incubation is not definitely known. Prodromes, such as malaise, irregular, fugitive pains, and sore throat (tonsillitis) may be present for a few hours or a day or two; but, as a rule, the

disease sets in abruptly with chilliness (occasionally a definite chill), fever, and inflammatory manifestations in one or several joints. The affected joints are usually the larger joints—the knee, ankle, shoulder, elbow, and wrist; less commonly the hip, fingers, toes, and intervertebral joints. They are swollen, red, hot, painful, and tender; motion is much restricted and when made aggravates the pain. A characteristic of the joint lesions is their fugaciousness, the fact that the inflammatory phenomena sometimes develop and subside with marked rapidity (but sometimes more slowly), and that they flit from one joint to another. In different cases, therefore, at one time, one or several joints may be involved in varying degrees. In some cases, later in the course of the disease, the lesions show a disposition to linger in certain joints. It is rare, however, for a single joint only to be involved. Intra-articular effusion is usually slight or moderate, the marked swelling of the joint being due largely to serous infiltration of the periarticular tissues—the tendon and muscular sheaths, bursæ, etc. Rheumatic nodules (misnamed fibrous nodules) are present in some cases, and are of much diagnostic importance. They are more common in children than in adults, and consist of small swellings (1 to 4 mm. in diameter) attached to bony prominences (the periosteum) or tendon sheaths just beneath the skin—the hands, wrists, elbows, vertebræ, etc. They are sometimes more apparent to sight than to touch, are usually not tender, and sometimes appear and disappear with marked rapidity, although they may last for a long time (when they really become fibrous nodules).

The general manifestations of rheumatic fever are usually subsidiary to the articular. The fever, as a rule, varies from 102° to 104° F., though hyperpyrexia (106° to 108° F.) is occasionally observed in severe cases. The fever pursues an irregular course and ends by lysis. The pulse is rapid (above 100) and soft; other symptoms of toxemia are present, such as loss of appetite, thirst, coated tongue, constipation, enlargement of the spleen, and concentrated urine (which is usually highly acid and may be albuminous). Profuse acid sweats of a peculiar odor are the rule and often correspond with remissions in the temperature; and sudamina and milaria are common. Leukocytosis is present; a secondary anemia usually develops rapidly.

Complications.—Complications are many, though doubtless many lesions heretofore regarded as complications are really part and parcel of the rheumatic fever (manifestations of the rheumatic poison); but some are the result of secondary infection. Hyperpyrexia (108°, even 110° F.) is occasionally observed, especially in primary attacks. It is a serious symptom, usually associated with severe nervous manifestations, such as delirium, rapid pulse, marked prostration, and frequently soon leads to death. The term "cerebral rheumatism" has been given to some cases in which toxic (nervous) symptoms are severe, usually from the beginning. To the high fever, delirium, and rapid pulse, evidences of motor irritability (tonic spasms, sometimes general convulsions) may be added; these are often followed by coma and death. The brain at

necropsy usually reveals no lesions; in some cases meningitis (purulent and other) has been found; but these cases were probably not true rheumatic fever, the joint lesions being pyemic and improperly interpreted clinically. Delirium and coma may occur without hyperpyrexia—being in some cases uremic. Chorea may occur in association with or as a sequel to rheumatic fever. Cardiac complications are the most common, and in many respects the most serious. Myocarditis (toxic) is common in the early stages and often leads to dilatation. Endocarditis occurs in many cases (50 to 75 per cent.), and its likelihood increases with the youth of the patient and the number of attacks. The lesions most frequently involve the mitral valve, and may be benign (usually) or malignant (unusually). Often the lesions are mild and overlooked, and are not detected until later in life, when sclerotic changes in the valve segments have occurred. Many of the murmurs that develop during rheumatic fever, however, are not attributable to endocarditis, but to dilatation of the heart or the very common anemia, and they disappear with convalescence. Pericarditis is not uncommon, and is found in most fatal cases. It may be fibrinous, serofibrinous, or purulent. In many cases the lesions constitute a pancarditis. Serous pleuritis and even serous peritonitis may also occur, and two or more serous membranes may be involved simultaneously—a multiple serositis. Congestion of the lung and pneumonitis are observed in some cases and may prove fatal. Tonsillitis and pharyngitis are sometimes marked; nephritis is rare. Different skin lesions have been observed—sudamina, miliaria (already mentioned), urticaria, multiform erythema, scarlatiniform rash, purpura, etc.; so-called purpura (peliosis) rheumatica is really not a rheumatic affection. The muscles are often swollen and painful, and sometimes become atrophic (arthritic atrophy).

Rheumatic fever in children usually sets in abruptly with high fever, and sometimes convulsions. It involves the joints, as a rule, less than the heart—myocarditis (with dilatation), endocarditis, pericarditis; anemia and exhaustion develop apace, and if the child does not soon die, the fatal termination from valvular disease, etc., is usually merely a question of time. Aberrant rheumatism is quite common in children—growing pains, stiff neck, recurring tonsillitis, etc.

Diagnosis.—The diagnosis is usually quite apparent from the fever, polyarthritis (and its peculiarities), acid sweats, and the rarity of involvement of the temporomaxillary, sternoclavicular, sacro-iliac, intravertebral, and pubic joints. Gonorrhœal arthritis may be distinguished by a history of gonorrhœa; involvement of one joint often (especially the knee or wrist), although several joints may be involved; severity of the local lesions—extreme pain and marked swelling due chiefly to periarticular œdema; persistence of the articular lesions—often leading to more or less permanent disability of the joint; relative absence of general symptoms—little fever, etc. Acute osteomyelitis, involving especially the lower end of the femur or the upper end of the tibia, may be distinguished by the involvement of the epiphysis and shaft of the bone (rather than the

articulating surfaces), the great intensity of the local signs, and the grave constitutional symptoms (pyococcic). Acute arthritis, such as may occur in infants, in scarlatina, in cerebrospinal meningitis, in puerperal infection, etc., is usually septic in character (pyococcic), may go on to suppuration, and is attended by the usual symptoms of septicemia—chills, fever, sweating, etc., and a focus of infection may often be demonstrable. If not pyococcic, the so-called acute rheumatic joints of children are likely to be syphilitic or scorbutic. Gout may be distinguished by its occurrence in males (especially beyond the fiftieth year), its characteristic onset, predilection for the great toe (though other joints are often involved), uratic deposits in the body, and an analysis of the urine.

Prognosis.—Rheumatic fever is very variable in its course, though it usually terminates in recovery without permanent damage to the joints in from two to six weeks. In some cases, however, recovery is less rapid, and exacerbations of fever and articular lesions recur from time to time—subacute rheumatic fever. The prognosis is worse in children than in adults, and in both is influenced by the occurrence of complications—some of which, such as hyperpyrexia and purulent pericarditis, are immediately serious, whereas others, such as endocarditis, are of more remote concern. One attack predisposes to others.

Treatment.—The patient should be confined to bed, even in the mild cases, and should wear a flannel night gown and sleep between blankets. The diet should consist largely of milk, but broths and soups may be allowed, and water should be given in large quantities. Salicylic acid and its derivatives exert a more or less specific action and should be given in full doses at the outset in all cases with the idea of obtaining an immediate effect. In a case of average severity in an adult sodium or ammonium salicylate, aspirin, salicin, salicylic acid, or other well-known salicyl compounds, should be administered in 20 grain (1.3 gram) doses, every two hours, until the symptoms abate or manifestations of salicyl intoxication supervene. By the third day usually the dose may be reduced to 15 grains (1 gram) every four or five hours. If symptoms of marked intoxication (tinnitus aurium, gastric irritability, delirium in some cases), supervene, the drug may be withdrawn for twelve to eighteen hours. Oil of wintergreen in 20 minim (1.25 c.c.) doses may be substituted for the other salicyl compounds. Should the attack not respond to the salicylates, the so-called alkaline treatment may be employed, or both may be used together—a form of treatment especially serviceable in children. To an adult 30 grains (2 grams) of potassium bicarbonate may be given every two or three hours for the first few days or until the urine is alkaline; or potassium acetate or citrate or sodium bicarbonate may be used. The salicyl treatment seems to relieve the joint symptoms, to shorten the course of the disease, to render relapse less likely, and to protect the heart, although endocarditis appears to be less common under the alkaline treatment. With a view to protect the heart, Caton urges the use of small blisters

applied successively along the course of the third, fourth, fifth, and sixth left intercostal nerves.

Attention to the local articular symptoms is of the utmost importance; but of all forms of treatment suggested, none is so valuable as absolute rest. Under the beneficent influence of rest enjoined by a splint the local pain and tenderness often subside almost immediately. In addition the joint may be wrapped in cotton wool, or in cloths saturated with lead water and laudanum, or a saturated solution of magnesium sulphate; or an ointment of methyl salicylate (20 per cent.) may be applied. Occasionally the pain is so severe as to call for the use of morphine, especially at night. In this event a Dover's powder is often serviceable. The coal tar products are scarcely ever necessary. Hyperpyrexia calls for the cold bath or the cold pack. Since anemia usually develops rapidly, iron is practically always indicated during convalescence, when quinine and strychnine will be found useful adjuvants.

PERTUSSIS.

(*Whooping Cough.*)

Pertussis is a subacute, specific, infectious, and moderately contagious disease, characterized by catarrhal inflammation of the respiratory mucous membrane and paroxysms of convulsive coughing that terminate in a protracted inspiration—the "whoop."

Etiology.—Pertussis is essentially a disease of childhood, being especially prevalent between the second and the seventh year. It is unusual under six months of age. It probably attacks the sexes equally, although girls are said to be more susceptible than boys. It sometimes occurs in non-immune adults, and even in elderly persons, in whom it usually runs a severe and frequently fatal course. In some cities the disease is endemic; in most sections of the country sporadic cases occur from time to time; but the disease usually prevails epidemically in the winter and spring. It frequently follows or precedes epidemics of measles, rarely epidemics of scarlatina. One attack usually establishes immunity, although recurrences are observed. Some persons possess a natural immunity.

The exciting cause of the disease is probably a bacillus, somewhat resembling the influenza bacillus, found by Koplik, and by Czaplewski and Hensel, in the mucous discharges. The disease is only moderately contagious, and especially during the catarrhal stage. Infection is usually transmitted directly, but it may be transmitted by a third person and perhaps by fomites. It is acquired through inhalation, and is somewhat favored by overcrowding, such as obtains in close, insanitary dwellings, school rooms, etc., and by previous ill health, especially catarrh of the air passages; but prolonged personal contact with an infected person seems necessary for infection.

Pathology.—Pertussis is devoid of characteristic lesions. The lesions found after death are those of the complications, such as broncho-

pneumonia, capillary bronchitis, atelectasis, emphysema, etc., but they present nothing characteristic.

Symptoms.—The period of incubation is from seven to ten days. Clinically the disease may be divided into two stages—the catarrhal and the paroxysmal. The invasion is rather slow, the disease beginning as an ordinary catarrh of the air passages, with injection of the conjunctivæ, coryza, cough, a few rales, and moderate fever (102° F.). Gradually the paroxysmal stage is developed. At the end of ten or fourteen days, the fever having almost if not entirely subsided, the cough, instead of improving (as in ordinary catarrh), gradually becomes extremely severe and paroxysmal in character, in some cases especially at night. A typical paroxysm consists of a series of short coughs, increasing in severity and interrupted only occasionally by a prolonged inspiration—the characteristic whoop. The child begins to cough, the expiratory efforts succeed one another with great rapidity until the lungs are almost emptied of air (sometimes they last even longer, consisting then of purposeless and almost noiseless vibrations of the chest wall), until finally with apparently a great effort a prolonged high-pitched crowing inspiration—the "whoop"—follows through the partially closed glottis. During the attack the child becomes more or less asphyxiated—the face becomes swollen and livid, the cervical veins swell, the conjunctivæ become injected, the eyes protrude and become bathed in profuse lacrymation, and subconjunctival and subdermal facial ecchymoses, and even hemorrhages from the nose, mouth, and sometimes the lungs, may occur. Usually the attack is terminated by the expectoration of a small amount of characteristic, tenacious, glairy mucus; frequently the attack terminates in vomiting, which serves to eject a considerable amount of tenacious mucus. The paroxysms may occur singly or several may follow in rapid succession, and they may be repeated several times or as often as forty or fifty times during the twenty-four hours. During the intervals, in the absence of complications, such as bronchopneumonia, the child may appear relatively well and may play. Spells of coughing devoid of the terror and not followed by the typical whooping spells may also occur. In adults the paroxysms are usually less severe and less typical than in children. A lymphocytic leukocytosis is present in about 85 per cent. of the cases, especially during the early (catarrhal) stage. The physical signs are those of bronchitis, and are not distinctive.

Having lasted three or four weeks, as a rule, the paroxysms gradually lessen in severity and frequency, expectoration becomes freer, mucopurulent in character, and gradually lessens (the stage of decline). The paroxysms, however, may last for weeks, even months, and emotion and excitement have been known to provoke an attack long after the disease has been apparently cured.

Complications and Sequels.—The most frequent and most important complication is bronchopneumonia, or capillary bronchitis, which is responsible for most of the fatalities. Emphysema sometimes arises in

consequence of the severe coughing. Usually it subsides, but it may form the basis of an emphysema that may give rise to symptoms in later life. Hemorrhage into the brain, occasioning convulsions, paralysis, even death, has occurred. Anemia and marked emaciation sometimes result from defective nourishment occasioned by the repeated vomiting. Ulceration of the frenum of the tongue sometimes results from forcible projection of the tongue against the teeth. Severe heart strain and its consequences, tuberculosis, swelling of the bronchial lymph nodes (sufficient to occasion substernal and vertebral dulness), nephritis, temporary glycosuria, noma, etc., have been observed as sequels.

Diagnosis.—The diagnosis in the early or catarrhal stage is impossible. As soon as the whoop appears the diagnosis is clear. In the absence of an epidemic, however, the disease may be suspected in the event of severe coughing (without the whoop) and vomiting, puffiness of the conjunctiva, or swelling and duskiness of the face.

Prognosis.—In consequence of the common association of complications, pertussis is a very fatal disease—the mortality in some epidemics being as high as 12 per cent. It is especially fatal in children under two years of age, and in the weakly.

Treatment.—The patient should be isolated and the ordinary measures of prophylaxis carried out (page 29). During the catarrhal stage rest in bed is essential, and the room should be well ventilated by day and by night. If two rooms are available, the child may be taken from one to the other, and each fumigated daily with a view to prevent re-infection. The diet should consist largely of milk, but after the catarrhal stage has passed other articles of a readily assimilable nature may be permitted—bearing in mind the frequent vomiting induced by the paroxysms of coughing. Sometimes it is wise to give a small amount of food immediately after the cessation of a paroxysm that has brought on vomiting. During the catarrhal stage a simple febrifuge or sedative expectorant mixture, such as is serviceable in an ordinary acute bronchitis, may prove of value. For treatment during the paroxysmal stage many remedies have been suggested from time to time, but none is really satisfactory. Quinine has long been used, and is probably as satisfactory as any drug—$\frac{1}{6}$ grain (0.01 gram) for each month of age, or 1.5 grain (0.09 gram) for each year of age under five years, three times daily. Jacobi has obtained the best results from the use of belladonna when given to the physiological limit. Many writers extol the virtues of antipyrin, 1 grain (0.06 gram) three times daily to a child of one year. Its ill effects may be somewhat neutralized by the concurrent use of whiskey; and it may be combined with the bromides or with belladonna; or belladonna may be combined with the bromides. Bromoform, asafœtida, and many other remedies have enjoyed a more or less ephemeral reputation. Local treatment is sometimes efficacious—sprays or swabs to the throat of hydrogen peroxide, resorcin (1 per cent.), and other antiseptics; or compound tincture of benzoin, creosote, carbolic acid, or oil of eucalyptus, by inhalation (croup kettle). Spirit of chloroform, or chloral, or rarely opium (paregoric,

Dover's powder, heroin, or morphine) may be resorted to in severe cases at night to insure rest. An elastic binder, as originally recommended by Kilmer, is of service in obviating vomiting, as well as in supporting the abdominal wall during severe paroxysms of coughing.

INFECTIOUS PAROTITIS.

(*Epidemic Parotitis; Mumps.*)

Infectious parotitis is an acute, infectious, and moderately contagious disease, characterized by inflammation of the parotid gland, and exhibiting a predilection to involve the testicles, the breasts, and the ovaries.

Etiology.—Infectious parotitis is most common in children, but it occurs also in adults; it is rare in infants and the aged. It is more common in males than in females. It usually prevails epidemically, especially in the late fall, the winter, and the early spring, but sporadic cases are not uncommon. The exciting cause of the disease has not been isolated. The disease is contagious, and though the contagium may be transmitted by fomites, rather close personal contact seems necessary for infection in most cases. The breath and the saliva are believed to be infectious—for about three weeks. Infection is probably acquired by inhalation and is transmitted along Stenson's duct.

Pathology.—The lesions consist of swelling of the mucosa of Stenson's duct, and hyperemia and œdema (sometimes minor inflammatory alterations) of the parotid gland and the periglandular tissues.

Symptoms.—The period of incubation varies from one to three weeks, and, as a rule, is unattended by prodromal symptoms. The onset is usually with moderate fever (101° to 102°, occasionally 103° to 104° F.), pain at the angle of the jaw, headache, and general malaise. Swelling of the parotid begins soon, and at the end of thirty-six or forty-eight hours is usually quite marked—occasioning swelling in front of and below the ear and elevating the ear. Sometimes the swelling and œdema extend far onto the cheek, into the infraparotid region, and the floor of the mouth. The region is painful (though rarely as painful as one might judge from the extreme swelling) and tender; opening the mouth, chewing, and swallowing cause distress; and usually there is ptyalism (though occasionally the mouth is very dry). In most cases the lesions are unilateral at first (most frequently left-sided), but in a day or two both glands are usually involved. Occasionally the other salivary glands are swollen; the spleen is usually enlarged and may be palpable. In a few cases there is quite high fever and marked prostration. The fever subsides by lysis, and by the seventh to tenth day the patient is usually convalescent. Suppuration is quite rare, and a second attack is uncommon (though not unknown).

Complications.—Orchitis is common after puberty, and may be single or bilateral. It usually develops as the parotid swelling subsides; rarely it precedes the parotitis. Atrophy not infrequently ensues in

severe cases. Vulvovaginitis is not uncommon in young girls; the breasts may become enlarged and painful; and rarely the ovaries are swollen and tender. Rarer complications comprise otitis media, meningitis, and divers nervous disorders (paralyses, etc.).

Diagnosis.—The diagnosis is usually easy. One should exclude disorders of the throat that might cause enlarged lymphatics, as well as dental caries with suppuration, etc.

Prognosis.—The prognosis is good; suppuration is rare, and a second attack uncommon.

Treatment.—Rest in bed is advisable. The bowels should be well opened with a saline cathartic. A simple diuretic fever mixture (aconite, potassium, citrate, etc.) is serviceable in many cases. An antiseptic mouth wash, hot fomentations, an ice-bag, or ichthyol ointment may be applied to the parotid swelling. In the event of high fever and delirium, the usual hydropathic and other treatment for such toxemic states is called for. Should orchitis develop, the patient must be put absolutely at rest, the testicles elevated, and lead water and laudanum, or some similar preparation, applied.

YELLOW FEVER.

Yellow fever is an acute, specific, infectious disease, characterized by fever, jaundice, albuminuria, and a disposition to hemorrhage, especially from the gastric mucous membrane.

Etiology.—Yellow fever is endemic in the West Indies and Central American sea ports; Havana was for more than a century a well-recognized focus of endemic infection, whence the disease was periodically widely disseminated. The spread of the disease is favored by general insanitary conditions, low lands, and the summer months—all of which are now known to bear an intimate relation to the mosquito. One attack confers immunity. The apparent immunity of natives in yellow fever zones is due largely to their having survived an attack of the disease during childhood.

Though Carlos Finlay, in 1881, stated that the mosquito is active in propagating yellow fever, the exact etiological factors remained unknown until the United States Army Commission, composed originally of Drs. Walter Reed, James Carroll, Jesse W. Lazear, and Aristides Agramonte, proved the correctness of Finlay's contention, and added much additional information—one of the members, Lazear, lost his life in the investigation; Reed and Carroll escaped a similar fate, but undermined their vitality, and later succumbed. The conclusions of the Commission are as follows: (1) The mosquito, Stegomyia fasciata, serves as the intermediate host for the parasite of yellow fever. (2) Yellow fever is transmitted to the non-immune individual by means of the bite of the mosquito that has previously fed on the blood of those sick with this disease. (3) An interval of about twelve days or more after contamination appears to be necessary before the mosquito is

capable of conveying the infection. (4) The bite of the mosquito at an earlier period after contamination does not appear to confer any immunity against a subsequent attack. (5) Yellow fever can also be experimentally produced by the subcutaneous injection of blood taken from the general circulation during the first and second days of this disease. (6) An attack of yellow fever, produced by the bite of the mosquito, confers immunity against a subsequent attack of the non-experimental form of this disease. (7) The period of incubation in thirteen cases of experimental yellow fever has varied from forty-one hours to five days and seventeen hours. (8) Yellow fever is not conveyed by fomites, and hence disinfection of clothing, bedding, or merchandise, supposedly contaminated by contact with those sick with this disease, is unnecessary. (9) A house may be said to be infected with yellow fever only when there are present within its walls contaminated mosquitoes capable of conveying the parasite of this disease. (10) The spread of yellow fever can be most effectually controlled by measures directed to the destruction of mosquitoes, and the protection of the sick against the bites of these insects. (11) While the mode of propagation of yellow fever has now been definitely determined, the specific cause of this disease remains to be discovered.

Stegomyia fasciata bites in the day time (which is quite characteristic); it is a very black mosquito with silver bands on its body and legs, a lyre-shaped silver mark on the back being characteristic. Although the specific microörganism of yellow fever has not yet been discovered, it passes through a fine porcelain filter, and doubtless therefore belongs to the ultramicroscopic organisms.

Pathology.—Jaundice is constant. The blood is disorganized, and hemoglobinemia may be present. Hemorrhages from the mucous membranes, especially the stomach, are quite characteristic; less frequently there are cutaneous and serous membrane hemorrhages. The liver, at first, is congested; later it is pale, and reveals fatty degeneration and focal necroses. The kidneys and the heart show parenchymatous degeneration. The lymph nodes are usually enlarged.

Symptoms.—The period of incubation is usually two to three days (forty-one hours to five days and seventeen hours, according to the United States Army Commission), and is usually unattended by prodromes, except perhaps slight headache and malaise. The onset of the disease is usually abrupt, with chill or chilliness, fever (103° to 104° F.), headache, severe pains in the eyes, the back, and the legs, epigastric distress, nausea, vomiting, coated tongue, and constipation. On the first day often a peculiar facies (one of the characteristic symptoms of the disease, according to Guiteras) develops—congested face, injected eyes, slight swelling of the eyelids and lips, and a noticeable element of jaundice ("undoubtedly the most characteristic feature of the facies," Guiteras). By the evening of the first day the temperature may reach 106° F. or more, but in the majority of cases it is scarcely more than 104° F.; it remains at this level with slight fluctuations for two or three days

and then gradually falls. In very mild cases the lysis may occur even on the second day. At the onset the pulse rate is somewhat increased, but it is rarely more than 100 or 110; on the second day it begins to fall, even with a rising temperature (a second characteristic symptom of the disease, Guiteras), and it may be not more than 75, with a temperature of 104° F. or more. The pulse becomes still slower during convalescence—60, 40, even less. Albuminuria is a third characteristic symptom, according to Guiteras; it varies much in intensity, but is usually more marked in the evening urine. In severe cases it may be present on the evening of the first day, and it is practically always present by the evening of the second day, being one of the few diseases in which albuminuria is present very early. It may be slight and transient, but is sometimes extreme and associated with tube casts and other manifestations of nephritis, and is usually disproportionate to the severity of the other symptoms. Complete suppression of urine may supervene and the patient may die of uremia even within thirty-six hours.

With the fall of the temperature, on the third day of the disease, the period of remission (so-called period of calm) develops; but this is often missed. In favorable cases the patient may now go on to complete recovery. Usually, however, he exhibits evidences of infection, such as jaundice, epigastric distress, albuminuria, mental dulness, and prostration, and at the end of twenty-four or thirty-six hours of comparative comfort (in severe cases, without such period) the period of secondary fever or collapse supervenes. The fever returns and becomes remittent; jaundice and albuminuria increase, and the urine may become almost if not quite suppressed; epigastric distress becomes worse; nausea and vomiting return, the vomitus consisting at first of the stomach contents, but soon of altered blood (black vomit); hemorrhages from the other mucous membranes and into the skin may develop; prostration becomes extreme, and the patient passes into a typhoid state (Hippocratic facies, coma, delirium, subsultus tendinum, etc.), and commonly dies. Recovery, however, may ensue even in the severest infections. In some cases the temperature does not become elevated, but may become even subnormal, "black vomit" occurs, the patient collapses, and dies (algid form). Mental alertness is a noteworthy feature of many cases, even in the presence of "black vomit," but delirium may occur at any time, even at the onset; when it occurs later, however, it is due quite as often to uremia as to the yellow fever intoxication. The bowels are usually constipated, and the stools are bile-stained (normal), but later they may contain blood.

Varieties.—Mild, severe, and fulminant cases may be distinguished. The mild cases often escape recognition, since the patient complains only of slight indisposition for a day or two, and does not take to bed. In certain (fulminant) cases the patient is overwhelmed with the intoxication; vomiting, collapse, coma, perhaps convulsions, develop, and the the patient dies within a day or two. Convalescence is often protracted, and attended with anemia, enteritis, abscesses, parotitis, etc.

Diagnosis.—The recognition of the disease is comparatively easy during the prevalence of an epidemic, but some epidemics have been so mild that much acrimonious discussion regarding their true nature, whether the disease is dengue or yellow fever, has arisen, especially in the South. In case of doubt, the three characteristic features of the disease mentioned by Guiteras should be of much importance. Guiteras also believes that a high hemoglobin value (90 to 100 or more) in the first four or five days of the disease is of diagnostic value—serving to exclude malaria, typhoid fever, and influenza, in which the hemoglobin is said to become low early in the disease. In dengue jaundice is not so common as in yellow fever, nor does it occur so early, and hemorrhages are less frequent. Pernicious malaria fever may be excluded by the absence of malarial parasites from the blood, absence of enlarged spleen, and the presence of the characteristic symptoms of yellow fever.

Prognosis.—The mortality varies in different epidemics between 15 and 85 per cent., and is in direct ratio to the severity of the infection and the resistance of the individual.

Treatment.—The patient should be quarantined (page 29), and, in accordance with what we now know of the etiology of the disease, especial attention should be directed to screening the patient's bed, room, or house (so as to prevent the access of mosquitoes), and to destroying all mosquitoes and their breeding places or places suitable for breeding. Special attention must be directed to the occurrence of mild cases, especially among children and young adults; these often act as sources of infection.

Since we have no specific, the treatment of the developed disease is largely supportive and symptomatic. The patient must rest in bed until he is entirely convalescent, and should have a plentiful supply of fresh air, sunshine, and light, nutritious food. Hot fomentations to the back and epigastrium, and an ice-bag to the head may relieve the pains; morphine and the coal-tar products should be given only in the very early stages of the disease, if at all, and very cautiously. Fever calls for hydropathic measures—which not only reduce the temperature, but act as a general nervous stimulant. Vomiting is often intractable, but may be controlled by cracked ice, sips of very hot water, cocaine, or dilute hydrocyanic acid. Large amounts of water should be given by the mouth (if retained), and saline solutions by the bowels or hypodermicly. Alkalies, such as sodium bicarbonate, may be added to the drinking water, to reduce the acidity of the gastric juice (as well as the urine); or one may use, as advised by Sternberg years ago, sodium bicarbonate, 4 grains (0.25 gram), mercuric bichloride, $\frac{1}{125}$ grain (0.0005 gram), and water, 2 teaspoonfuls (10 c.c.) every hour in a severe case. Other intestinal antiseptics may be tried, such as salol, naphthalene, etc. Quinine, as well as iron, has been highly recommended, especially to control the hemorrhages, over which, however, we can exert very little influence. Stimulants are usually called for, sooner or later—strychnine, alcohol, etc. Uremia is to be treated on general principles.

DENGUE.

(*Break-bone Fever; Dandy Fever.*)

Dengue is an acute, specific, infectious, and highly contagious disease, characterized by high fever, severe neuromuscular and arthritic pains, and an initial erythematous and a subsequent polymorphous rash.

Etiology.—Dengue prevails endemically, even pandemically, in warm climates, such as India, Cairo (and the surrounding country), Java, the West Indies, our own Southern States, etc., and during the summer months sometimes spreads to the subtropical and temperate zones. The disease is highly infectious, and once developed attacks almost the entire population almost simultaneously—no age, sex, nor race escaping. One attack does not confer immunity.

The exciting cause of the disease has not been isolated with certainty; it is probably ultramicroscopic. Graham, in Beirut, Syria, found what he believes to be a hematozoön in the blood, and believes the mosquito, Culex fastigans, to be the active factor in propagating it. Ashburn and Craig also inculpate the mosquito.

Pathology.—Since the disease is rarely fatal, careful and extended pathological studies have not been carried out.

Symptoms.—The period of incubation is from one to five days, and is unattended by prodromal symptoms. The onset is sudden, with chill or chilliness, fever (104° to 107° F.), and severe, even excruciating, pains in the head, eyeballs, back, and the extremities (the muscles and joints)—whence the term break-bone fever. The large or small joints, and few or many, may be affected simultaneously or successively, and in addition to being painful, they may be also red and swollen. Furthermore, the pulse is rapid, the appetite lost, the urine concentrated, and the patient may be slightly delirious; the face is bloated, the eyes injected, and the skin and the visible mucous membranes may exhibit a diffuse erythematous rash, and the skin may be unduly sensitive. On the third or the fourth day the fever falls usually by crisis attended by sweating, sometimes by lysis, and, aside from some muscular soreness and stiffness, the patient feels comparatively well. At the end of two or three days of apyrexia, the fever and the neuromuscular pains return, but they are usually less severe and last not more than twenty-four to forty-eight hours; they are often accompanied by a polymorphous rash—measly, scarlatiniform, papular, sometimes urticarial and even vesicular—which develops first upon the hands, and then spreads to the arms, face, trunk, and legs. This gradually fades and is followed by a fine, branny, often almost imperceptible, desquamation. The lymph nodes are often enlarged, and may remain palpable well into convalescence or beyond. Hemorrhages from the mucous membranes have occasionally been observed.

The disease lasts usually from seven to ten days, but convalescence

is sometimes protracted by prostration, anemia, insomnia, even meningitis, or a relapse.

Diagnosis.—The epidemic prevalence of the disease, the almost simultaneous implication of a very large portion of the population, the characteristic fever curve, and the cutaneous rashes serve to distinguish the disease, although some difficulty may be experienced in differentiating rheumatism in early cases, and influenza, measles, and scarlatina may have to be taken into consideration in other cases. Greatest difficulty has been experienced in differentiating yellow fever, but yellow fever is suggested by the characteristic jaundiced facies, the slowing of the pulse with increasing temperature, the early albuminuria, and by the fact that slight jaundice, though it may be observed occasionally in dengue, does not occur as early as in yellow fever, and hemorrhages are much less common.

Prognosis.—The prognosis is good. Death rarely results, and then only in the debilitated, and in the event of coma or hemorrhages.

Treatment.—The treatment is purely symptomatic. The severe pains usually necessitate the use of morphine, although quinine, salol, and other salicylates have proved of some service. Tincture of gelsemium has been highly recommended by McLaughlin. Chloral and the bromides are useful for the insomnia and general restlessness. High fever calls for the use of hydrotherapy. Tonics are required during convalescence, and potassium iodide is said to be valuable for the persistent arthritic pains.

HYDROPHOBIA.

(*Rabies; Lyssa.*)

Hydrophobia is an acute, specific, infectious disease communicable to man by the bite of an animal infected with rabies.

Etiology.—Rabies is a disease of dogs especially, but occurs also in wolves, foxes, cats, rarely in cows and horses, and may be communicated to pigs, rabbits, etc. The disease occurs in all parts of the world, and is probably more common in spring than in summer (contrary to the vulgar opinion). The etiological factor has not yet been isolated, but it exists in the saliva, in the brain, spinal cord, and nerves, and probably in other secretions, and the blood; it seems not to be present in the viscera. It is filtrable and doubtless belongs to the class of ultramicroscopic organisms. Infection is transmitted to men usually through the bite of a rabid dog, but not more than 10 per cent. of those bitten become infected. Children are more commonly affected, because they are more frequently bitten. Punctured wounds of the exposed surfaces, hands, face, neck, etc., are especially dangerous, clothes largely preventing wounds of other parts of the body from becoming infected.

Pathology.—The lesions consist of congestion of the medulla and the spinal cord, sometimes associated with minute hemorrhages, and are in no way specific—occurring in other convulsive states. Accumulations of

leukocytes about the bloodvessels and nerve cells, especially the motor cells of the medulla (Babes' rabic tubercle), are usually present. Van Gehuchten and Nélis have described proliferation of the endothelial cells of the capsules of the intervertebral, pneumogastric, and sympathetic ganglia, with destruction of the proper nerve cells. Negri has described protozoan-like bodies in the nerve cells, especially those in the horn of Ammon, in rabid animals. These lesions may be reproduced experimentally and are resorted to for diagnostic purposes.

Symptoms in the Dog.—Two forms of rabies are recognized: (1) Maniacal rabies, in which there is usually a preliminary stage of depression, followed by a stage of excitement or mania, in which the dog eats little, runs about with saliva drooling, and shows a disposition to bite persons, and real and imaginary objects; followed by a third or paralytic stage, in which weakness and emaciation proceed rapidly. Death ensues usually within eight days. (2) Dumb rabies, in which the second or excited stage is absent, and in which paralysis and death occur earlier. It is quite important to recognize this form, as, though more readily overlooked, it is quite as infectious as the maniacal form, and is the only form that occurs in rodents.

Symptoms in Man.—In man the period of incubation varies between two weeks and eighteen months; in most cases it is about eight weeks; and in children it is less than in adults. As a rule, the wound heals without noteworthy manifestations. The onset of the disease is insidious. Toward the end of the period of incubation the site of the wound may itch or pain a little, and the adjacent lymph nodes may be somewhat enlarged and tender, but often there are no local manifestations. The premonitory symptoms, which last one or two days, are depressive—melancholia, undue anxiety, loss of appetite; later, perhaps headache, irritability of temper, and intolerance for noises, and sleeplessness. The temperature may be slightly elevated and the pulse somewhat increased in frequency. Gradually spasm of the muscles of deglutition and of respiration, and a disposition to refuse water (hydrophobia) is developed. The stage of excitement then comes on apace. The spasm of the muscles of deglutition and of respiration become more marked; they become convulsive in character, and may even spread to the trunk and the extremities. They may be excited by the slightest touch, sound, visual impression, or auditory suggestion; and the paroxysms induced by the sight of water or the attempt to swallow are merely one expression of the universal hyperesthesia, of which the greatly exaggerated reflexes are another. The paroxsyms, however induced, may last for a few minutes or a half hour; they are associated with inability to swallow, sensations of extreme dyspnœa (even in the absence of obstruction to the air passages), and with the greatest mental anxiety, distress, and terror. Thirst is sometimes present, and the temperature is increased during the paroxysms. The inability to swallow the saliva (which is not infrequently increased), a slight spasmodic cough (designed to expel the saliva), and certain guttural sounds produced by spasmodic

movements of the larynx, occasion the "frothing at the mouth" and the "barks like a dog" associated in the public mind with the disease. Between the paroxysms the patient is mentally alert, and only too vividly apprehends his danger. Should the patient not die from asphyxia or exhaustion, in from two to four days the final, or paralytic, stage sets in; the spasmodic attacks lessen and finally cease, the heart becomes weak, exhaustion and coma supervene, and in from twelve to twenty-four hours the patient dies.

Diagnosis.—Under no circumstances should the suspected animal be killed, except by a physician with a view to secure the nervous tissues, examine them, and inoculate them into rabbits—which should always be done in animals suspected of rabies. In the event of rabies the inoculated rabbit dies of the paralytic form of the disease in from fifteen to twenty days; whereupon the lesions described by Van Gehuchten and Nélis may be found in the plexiform ganglion of the pneumogastric nerve, and the Negri bodies may be found in the horn of Ammon. Hydrophobia rather than tetanus is suggested by the history of a bite, the special involvement of the muscles of deglutition and of respiration, the psychic symptoms, and the absence of lockjaw, of opisthotonos, and of persistent tetanic spasm. Pseudohydrophobia or lyssaphobia is a neurosis that sometimes develops in hysterical and neurotic subjects, not infrequently as early as twenty-four hours after a bite by a rabid or a supposedly rabid dog, and often simulates true hydrophobia; usually, however, the patient has exhibited considerable dread lest hydrophobia develop, and the hysterical element is always in the foreground; the premonitory symptoms are missed, there is no true respiratory spasm, obvious imitations of a dog (such as bites, growls, etc.) are conspicuous, the disorder lasts longer than true hydrophobia, and is quite amenable to certain severe and well-recognized measures used to control hysterical attacks.

Prognosis.—No patient has been known to recover from an indubitable attack of hydrophobia.

Treatment.—If possible the wound should be excised; at least it should be well laid open and cauterized with nitric acid, carbolic acid, or caustic potash, and subsequently treated antiseptically as an open wound (not allowed to heal) for several weeks. Pasteur's preventive inoculations should be instituted as soon as possible. Pasteur found that the virulence of the rabic virus could be markedly increased by passing it through a series of rabbits (subdural injections of emulsions of the dog's spinal cord), so that ultimately the period of incubation is reduced to about six days, and the duration of the disease to between two and five days. The spinal cords of these rabbits contains his *virus fixe*, which, however, gradually loses its virulence upon being dried in the air, and gradually becomes inert. Beginning then, with the inoculations of least active virus and progressing to those of high activity, immunity in man is established (within about two weeks) before the period of incubation of the disease occasioned by the dog's bite has elapsed. By this mode of treatment, the mortality of an otherwise absolutely fatal disease has

been reduced almost to a minimum. Aside from the foregoing, treatment is merely palliative. When the disease has developed, the patient should be confined to a darkened room, free from noises; he should be nourished by the rectum, and his distress mitigated by chloroform, chloral, morphine, and the bromides. One may try the Buisson treatment, consisting of vapor baths and jaborandi, to induce profuse sweating; but the treatment has not commended itself to the thinking profession. An efficacious antirabic serum has not yet been produced, nor are Pasteur's preventive inoculations of the slightest service after the disease has developed.

ROCKY MOUNTAIN SPOTTED FEVER.

(*Tick Fever.*)

Rocky Mountain spotted fever is an acute, infectious disease, occurring in Western Montana, chiefly in the Bitter Root Valley, as well as in Idaho, Wyoming, and Nevada, and characterized by chills, fever, neuromuscular pains, and a peculiar maculopapular or petechial eruption.

Etiology.—Rocky Mountain spotted fever is transmitted by the bite of a tick, Dermacentor venustus (andersoni) in Montana, and by Dermacentor modestus in Idaho; and is transmissible to the monkey and the guinea-pig. The nature of the infectious agent is not known. Wilson and Chowning described a small parasite (resembling hyaline malarial bodies) which they found in the erythrocytes as well as free in the blood plasma, and which they have named Pyroplasma hominis (whence the name for the fever, Pyroplasmosis hominis). Stiles and Craig, however, doubt the identity of these supposed parasites, Craig stating that the appearances are due to changes in the hemoglobin. Ricketts has described a bacillus found in the tick and their fresh eggs.

Symptoms.—The period of incubation is said to be from three to twelve days. The onset is rather sudden, with chills, headache, nausea, neuromuscular pains, and fever. Epistaxis is common, the bowels are usually constipated, and there are manifestations of catarrhal inflammation of the conjunctiva and the upper respiratory tract. The pulse is usually disproportionately rapid—120 to 140. On the second to the seventh day of the disease a rather profuse maculopapular (occasionally petechial) rash appears. It is usually most marked on the wrists, arms, legs, forehead, and later on the trunk. Albuminuria is present in all cases. The fever reaches its height (103° to 105° F.) on about the twelfth day, and then falls by lysis to reach the normal in four or five days or a week. Concurrent with the fall in temperature the rash fades, and is sometimes followed by desquamation. Gangrene of the fingers, toes, and scrotum has been observed.

Diagnosis.—The disease is readily recognized in the section where it abounds, a history of a tick bite being important. From purpura the affection is distinguished by the absence of joint symptoms and of sore throat, and the early appearance of the rash, especially on the wrist

(as contrasted with its later appearance, especially on the trunk in purpura).

Prognosis.—The mortality is very high in some regions—from 70 to 90 per cent., death occurring usually on about the tenth day; in Idaho it is low—2 to 5 per cent.

Treatment.—Quinine seems to exert a more or less specific action, and should be given in large doses (15 grains, 1 gram, hypodermicly, every six hours, Anderson) throughout the course of the disease. Otherwise, the disease should be treated as typhoid fever. Stimulants are usually called for.

EPIDEMIC POLIOMYELITIS.

(*Acute Poliomyelitis; Infantile Paralysis.*)

Epidemic poliomyelitis is an acute, infectious, and mildly contagious disease, characterized by hemorrhagic inflammation and softening of the brain and spinal cord, often widespread, but sometimes limited to the anterior horns of the spinal cord; clinically it is characterized by fever and flaccid paralysis of one or more limbs.

Etiology.—Poliomyelitis may occur sporadically or epidemically. Sporadic cases occur widely distributed from time to time, but especially during the summer; they are almost exclusively confined in children from two to four years of age, although they do occur in older children. The epidemics have been most frequent in this country and in Norway. They have been more common in rural districts, but have occurred also in cities, such as New York, Christiania, Stockholm, etc.; they have prevailed especially during the autumn; and while many children have been affected, the disease has been quite common in young adults.

The exciting cause of the disease has not been isolated. Flexner and Lewis, using an emulsion of the spinal cord from a fatal case in a child, have been enabled to transmit the infection to monkeys, and using the spinal cords of infected monkeys to transmit the infection to several generations of monkeys. Infections have been made successfully by intracerebral inoculation, and by inoculation into a large nerve (sciatic), into the circulation, into the peritoneum, and beneath the skin. The virus is filtrable (like that of rabies and vaccinia), and is believed to belong to the class of ultramicroscopic organisms. How the infection is acquired is not definitely known, although it is supposed to be by inhalation, the naso-pharynx being the primary site of infection.

Pathology.—The lesions consist of inflammation, softening, and hemorrhage, in mild cases, more or less limited to the anterior horns of the spinal cord, and leading to marked degeneration of the motor ganglion cells; the lesions, even in mild cases, however, may be more widespread and may be associated with lymphocytic infiltration of the pia-arachnoid. Analogous lesions may occur in the medulla, pons, and gray matter of the cerebrum. In severe cases the lesions are those of diffuse myelitis and encephalitis, with more or less meningitis. Flexner and Lewis

have found the virus in the central nervous system, the cerebrospinal fluid, and mesenteric lymph nodes. After the disease has run its course, the affected portion of the cord is found lessened in size; the anterior horns are greatly atrophied and contain few if any ganglion cells; the corresponding motor nerve roots are atrophied; and the muscles supplied thereby are atrophic, sclerotic, and perhaps fatty. The lower motor neuron, therefore, bears the brunt of the infection.

Symptoms.—The period of incubation is not known. There may be slight prodromal symptoms, but, as a rule, the onset is abrupt, with fever (101° to 103° F.), headache, malaise, perhaps vomiting, and rarely convulsions. In some cases pains are complained of in the arms, legs, or back; but, as a rule, attention is first directed to the limbs by paralyses. This develops rapidly, usually within twenty-four hours; not infrequently the child, perhaps a little feverish on retiring for the night, is found paralyzed in the morning. The paralysis is, as a rule, at its maximum at the beginning, that is, usually it does not progress; it is irregular and asymmetrical in its distribution; it may involve a part of one limb, the entire limb, two or more, or all four limbs. In the legs the tibialis anticus and the extensors are most commonly involved; in the arm, there is an upper-arm type (deltoid, biceps, brachialis anticus, and supinator longus), and a lower-arm type (extensors or flexors of the wrist and fingers) —all of which is due to the group representation of muscles in the spinal cord. The muscles of the face (cranial nerves), the sphincters, and sensation are rarely affected. The reflexes are absent. The muscles supplied by the affected neurons soon undergo atrophy, become soft and flabby, and at the end of a week usually reveal the reactions of degeneration. As a rule, after several weeks, some improvement takes place; in case there was only slight involvement of a limb, almost complete recovery may ensue; but complete initial paralysis always results in more or less permanent disability. The limb remains smaller and shorter than its fellow, that is, growth is retarded; the muscles remain soft and flabby, and the skin cold and livid; and contractures and deformities may ensue. In the case the leg is involved, apparatus are often required to enable the patient to get about.

Diagnosis.—The diagnosis is readily made, fever and the rapid onset of flaccid paralysis in one or more limbs, soon followed by the reactions of degeneration, being unequivocal; these exclude the cerebral paralyses. The pseudoparalysis of infantile scurvy (acute rickets, scurvy-rickets) may be excluded by the usually complete loss of power and rapid atrophy; in infantile scurvy, movement is possible although it is painful, and there are the characteristic changes in the bones, cutaneous hyperesthesia, etc.

Prognosis.—The outlook is not hopeful; that is, complete restoration of structure and function is unlikely. The condition is usually at its worst in the first day or two; after a stationary period more or less improvement follows, but shortening, contractures, and deformities are common.

Treatment.—If seen during the period of fever, the child should be put to bed and given a diuretic and diaphoretic fever mixture (spirit of

nitrous ether, solution of ammonium acetate, or of potassium citrate, etc.). The bowels should be freely opened; and the affected limb or limbs may be wrapped in cotton wool. Starr recommends the use of hexamethylenamin, 1 grain (0.06 gram) every six hours, to a child three years of age; the drug is eliminated in the cerebrospinal tract and perhaps exerts some antiseptic action on the infectious microörganisms. Ergot, belladonna, quinine, the salicylates, and potassium iodide have also been recommended. Attention must be paid to the general nutrition of the child—good food and fresh air. As soon as the sensitiveness has subsided, rubbing, kneading, and warm baths to the affected limbs should be instituted; electricity is of less value, but may be employed. Strychnine is a serviceable remedy in this stage, and may be combined with other tonics. Deformities should be prevented by exercises and the use of orthopedic apparatus; developed deformities should be corrected, by operation if necessary.

ACUTE INFECTIOUS JAUNDICE.

(*Weil's Disease.*)

From the large group of cases of infectious or toxic jaundice, a special form, known as Weil's disease, is usually separated, but the propriety of this is doubtful. Described by Weil, in 1886, it is characterized by fever, jaundice, enlargement of the liver and the spleen, and nephritis. It prevails especially during the summer months, is most common in males between the ages of twenty and forty years, and seems to have a special predilection for butchers, soldiers, etc. It has been ascribed to Bacillus proteus fluorescens, which has been found in decaying meat, sausages, drinking water, etc. Some French cases are ascribed to Bacillus coli infections; others may be proteus infections. Doubtless all cases are not alike. Infection is believed to be acquired by eating infected meat or cheese, or drinking tainted water.

Pathology.—The tissues are bile-stained; the liver is normal in size or enlarged, the kidneys are swollen, and both organs exhibit fatty degeneration of the epithelium, and the liver focal necroses; the spleen is enlarged; and hemorrhages into the skin and the mucous and the serous membranes may be encountered.

Symptoms.—The onset is sudden, with chilliness, fever (103° to 104°F.), headache, malaise, rapid pulse, and severe neuromuscular pains, especially in the back and the calves of the legs, but not infrequently also in the arms and even the cheeks. Jaundice develops on the second day; it is usually slight or moderate in degree, but may become severe; it lasts ten days or more, and gradually lessens. The liver is usually swollen and tender, the spleen is enlarged; the stools may be light colored, but commonly contain some bile; and the urine contains albumin and casts. Delirium and the typhoid state may supervene. With the onset of the jaundice the pulse becomes slower. Gastro-intestinal symptoms are

unusual. The fever, which is characterized by marked remissions, lasts a week or ten days, and gradually subsides. Recovery usually ensues, but convalescence may be slow and relapses are not infrequent (40 per cent. of the cases, Quincke).

Diagnosis.—In the diagnosis, other forms of infectious or toxic jaundice, catarrhal jaundice, and bilious remittent fever must be considered.

Treatment.—The treatment is altogether symptomatic, and consists of rest in bed, a milk diet, cleansing of the intestine by calomel, the use of intestinal antiseptics, and the meeting of such other symptomatic indications as they may arise.

FOOT-AND-MOUTH DISEASE.

(*Epidemic Stomatitis; Aphthous Fever; Epizoötic Aphtha.*)

Foot-and-mouth disease, or epidemic stomatitis, is an acute, specific, infectious, and highly contagious disease of lower animals, transmissible to man, and characterized by a vesicular eruption on the oral mucous membrane and sometimes on the skin.

Etiology.—Epidemic stomatitis is common among cattle, sheep, and pigs, and is transmissible to the domestic animals, and rarely to man, through the medium of infected milk, butter, or cheese; rarely infection occurs directly through a wound (hands, arm, foot). In cows the udders are not infrequently involved, whence the ready transference of the disease through the medium of the milk. The specific etiological factor has not yet been isolated. Löffler, who has been able to vaccinate animals against the disease, believes that the specific cause is an ultramicroscopic organism, since it passes through porcelain filters the pores of which do not permit the passage of the smallest bacteria known.

Symptoms.—The period of incubation is from three to seven days. The onset of the disease is announced by moderate fever, slight chilliness, a sensation of heat in the mouth and throat, pain and difficulty in swallowing, and soon the eruption of vesicles on the lips, oral, buccal, and pharyngeal mucous membrane, occasionally also on the skin, especially the hands (about the nails), the feet (between the toes), and rarely elsewhere. At the end of several days the vesicles rupture, and leave small ulcers that usually heal in a week or ten days. Rarely the exanthem is widespread, and has even been known to be hemorrhagic. Severe gastro-intestinal symptoms and endocarditis may occur as complications. The duration of the disease is two to four weeks.

Diagnosis.—The diagnosis is easy; only the careless would mistake it for varicella.

Treatment.—The treatment is that of stomatitis. If it be necessary to use the milk during the prevalence of an epidemic among the cows, it should at least be boiled.

MILIARY FEVER.

(*Sweating Sickness.*)

Miliary fever is an acute infectious and moderately contagious disease characterized by fever, profuse sweats, and a papulovesicular rash.

Etiology.—The disease attacks children and adults apparently indiscriminately. It usually prevails epidemically—in Europe; as far as is known, no epidemic has occurred in this country. The exciting cause of the disease is not known. How the contagium is spread is not definitely known, but the large number of persons affected at the one time lends support to the view of aërial convection.

Symptoms.—The period of incubation is not definitely known, but it is probably not more than a day or two—during which there may be minor prodromal manifestations. As a rule, the onset is rather sudden, with fever and profuse sweating. The fever is usually not more than 103° F., but hyperpyrexia has been observed. In addition, the patient complains of headache, respiratory oppression (suggesting suffocation), anorexia, and constipation. On the second or third day the characteristic rash appears—on the skin (often first on the face) and the oral mucous membrane; it consists of minute, reddish papules, most of which soon develop a tiny vesicle at their apex—much resembling sudamina. Sudamina, that is, vesicles not on an inflammatory base, may also be observed. Soon after vesiculation, dessication, followed by desquamation, sets in. Sweating is constant, but exacerbations occur. In cases with high fever, other evidences of toxemia are present (delirium, prostration, etc.), and emaciation usually progresses rapidly. In favorable cases the fever falls by lysis, and by the tenth day the patient is convalescent. In unfavorable cases the patient dies with manifestations of increasing intoxication—delirium, coma, convulsions, hemorrhages, etc. In certain so-called fulminant cases death has occurred within the first few hours.

Diagnosis.—In an epidemic the disease is unmistakable.

Prognosis.—The mortality varies up to 25 per cent. in different epidemics. Relapses are common.

Treatment.—The treatment should consist of rest in bed, soft diet with a considerable amount of fluids (to replace the water lost by the sweats), hydrotherapy for its stimulating effect and to control the fever and toxemia, and stimulants (alcohol, strychnine, etc.); atropine may be indicated for the sweats.

MILK SICKNESS.

(*Trembles.*)

Milk sickness is an acute infectious disease of cattle ("trembles"), communicable to man, and characterized by gastro-intestinal irritation and nervous phenomena attributable to toxemia.

Etiology.—Trembles prevails among cattle, in the South and West, especially in newly settled regions, and is communicable to man and the young of horses and sheep. The specific infectious agent has not been isolated with certainty, although Jordan and Harris describe a so-called Bacillus lactimorbi. The infection is transmitted to man by means of milk, butter, and cheese; and these, as well as the flesh of infected cows, even after having been boiled, are poisonous to dogs.

Symptoms.—In cows, mild cases may be symptomless, unless the animal is hard-driven, although the milk may be infectious; in severe cases the cow refuses food, the eyes become injected, the animal staggers and trembles all over, and may soon die, often in convulsions. In man the symptoms are gastro-intestinal, such as a swollen, coated, and tremulous tongue, fetid breath, thirst, epigastric distress and pain, nausea, vomiting and constipation; and toxic, such as fever, restlessness, coma and convulsions, or a typhoid state. Death may occur in two or three days in acute fulminant cases. In other cases the disease may last from ten days to about three weeks, and be followed by a tedious convalescence.

Treatment.—The treatment is purely symptomatic.

BERIBERI.

(*Kakke; Endemic Multiple Neuritis.*)

Beriberi is a specific, probably infectious or toxic, disease of tropical and subtropical countries characterized by multiple neuritis and different consequent motor and sensory paralyses, muscular atrophy, and anasarca.

Etiology.—Beriberi is endemic in the Malay Archipelago, China, Japan, India, certain parts of South America (notably Brazil), the West Indies, the Dutch East Indies, the Philippines, and other South Pacific Islands. It is also occasionally met in different parts of the world, but largely as an imported infection. In this country it has prevailed especially among the Cape Cod fishermen, in asylums in Alabama and Arkansas, and in Louisiana. Bad hygienic surroundings, especially overcrowding, seem important etiologically, whence the frequent occurrence of the disease in barracks, jails, ships, etc.; but marked heat and humidity seem also to be of significance, whence its frequent occurrence during the rainy season. The yellow races are particularly susceptible. Males are more frequently affected than females, largely because of extraneous circumstances. The disease is most common during adolescence and early adult life—the fifteenth to the fortieth year.

The exciting cause of the disease has not been determined. There is considerable, though not conclusive, evidence that it is due to microorganismal infection, as maintained by Baelz, Scheube, and Wright, the last mentioned of whom believes that the infecting organism gains entrance to the body through the mouth and elaborates its toxin in the stomach and duodenum. Okata and Kokubo have described a coccus which they have isolated from the urine and which they believe is the

specific cause. The Japanese generally attribute the disease to bad food, especially rice and fish, which either itself directly causes the disease (whence a nutritional disorder due largely to deficient nitrogen supply or assimilation) or acts as the carrier of the infection or toxin. Ross attributes it to arsenical poisoning, since arsenic is a common adulterant of tobacco in the East, and he has found arsenic in the hair of a number of patients.

Pathology.—The characteristic lesions consist of degeneration of the peripheral nerves, involving the axons and medullary sheaths. In severe cases, similar lesions are found in the pneumogastric and the phrenic nerves. There is also congestion of the pylorus of the stomach and of the duodenum (considered specific by Wright), hyperemic and cloudy swelling of the liver and the kidneys, enlargement of the spleen, and parenchymatous and fatty degeneration of the cardiac and the voluntary muscles; and in some cases œdema of the subcutaneous tissues and serous cavities.

Symptoms.—The period of incubation is not known, but it has been said by Wright to be between ten and fifteen days. Clinically, Scheube distinguishes four types of the disease—largely variations in degree:

The *rudimentary form* begins usually with pain and weakness in the extremities, associated with paresthesia and anesthesia of the leg, frequently quite circumscribed and irregular in distribution. Slight œdema also may develop, the muscles may become tender, and palpitation of the heart, dyspnœa, and abdominal uneasiness may be observed. The symptoms last for a few days to several months, and generally disappear. Recurrences, however, are often encountered.

The *paralytic* or *atrophic form* presents, as a rule, symptoms similar to the foregoing, but the pains in the limbs are more pronounced, and the muscles of the legs soon become paralyzed and atrophic. The paralyses and atrophy may extend to the muscles of the trunk, less frequently to the arms, head, and neck. The paralyses are usually flaccid, reactions of degeneration are present, and the tendon reflexes are absent. The areas of anesthesia and of paresthesia are often irregularly distributed. The cardiac symptoms are not pronounced, and the œdema is usually slight (whence the term, dry beriberi). Recovery, when it ensues, is very slow.

The *dropsical form* begins as the preceding forms, but œdema soon appears, and the patient, as a rule, speedily becomes anasarcous—effusions into the subcutaneous tissues and the serous cavities. Palpitation of the heart, rapid heart action, dyspnœa, and cyanosis of the extremities are quite marked. Paralyses and muscular atrophy are not as pronounced as in the other cases, but even notable atrophy is not infrequently obscured by the existing œdema. The urine is diminished in amount, but, as a rule, contains no albumin. The condition persists for weeks and even months, and if recovery eventually ensues there is sometimes a very rapid disappearance of the œdema, with great increase in the amount of urine.

The *acute pernicious* or *cardiac form* begins as the preceding forms, but cardiac weakness is soon manifest, and complete cardiac failure may supervene within twenty-four hours. Usually, however, it lasts several weeks.

Diagnosis.—The disease where it is endemic presents no diagnostic difficulties, especially when several cases exist in the same vicinity. In sporadic cases, evidences of peripheral neuritis, with anasarca, and an irritable and weak heart, in a patient just arrived from the tropics is extremely significant. Arsenical and alcoholic neuritis should be excluded.

Prognosis.—The mortality varies from 2 per cent. in mild cases to 40 per cent. in severe cases. Patients seemingly progressing favorably not infrequently die of acute cardiac failure.

Treatment.—The patient should be removed from the endemic locality, be placed amidst the best hygienic surroundings, and given a diet rich in nitrogen and phosphorus. The treatment otherwise is largely symptomatic. Stimulants are valuable for cardiac weakness. Acute cardiac failure should be met with bleeding and free purgation. During convalescence the neuritis is to be treated as any other neuritis. Baelz has recommended the salicylates in the early stages of the disease.

EPIDEMIC DROPSY.

Epidemic dropsy is a subacute, infectious, and contagious disease characterized by fever, vomiting, diarrhœa, and the sudden appearance of anasarca.

Etiology.—Epidemic dropsy is peculiar to India, where it prevails especially during the cold weather, and attacks adolescents and adults of both sexes. It is rare in infants. The exciting cause of the disease has not been isolated, though there is some evidence that the contagium is more or less readily communicable from the sick to the well.

Symptoms.—The onset of the disease is often marked by fever, varying from 99° to 102° F., which often precedes but sometimes accompanies the development of dropsy—the most characteristic feature of the disease. Vomiting and diarrhœa are common, and sometimes mark the development of the disease. The dropsy begins, as a rule, in the legs, and soon involves the entire body; it may be preceded by burning, pricking, and itching of the skin. Marked anemia soon supervenes, and disturbances of circulation and respiration are prominent in almost all cases. An erythematous rash often appears on the face and trunk about a week after the development of the dropsy. The disease runs its course in from three to six weeks. The mortality varies from 2 to 40 per cent., and death results from œdema of the lungs, hydrothorax, hydropericardium, or other cardiac or pulmonary complications.

Treatment.—The treatment is purely symptomatic.

NASHA FEVER.

(*Nakra Fever.*)

Nasha fever is an acute infectious disease, peculiar to certain parts of India, especially Bengal, and characterized by fever, swelling of the nasal mucous membrane, and sometimes coma and death.

Etiology.—The nature of the disease is not known. Exposure, unhygienic conditions, and poor food seem to predispose to it. Women apparently are more predisposed than men; children and the aged are relatively immune. It prevails mostly from April to August. One attack seems to predispose to others, which sometimes develop at more or less definite intervals. The disease is believed by some observers to be a manifestation of malaria, but this is doubtful, since the malarial parasite has not been found and the disease is refractory to quinine.

Symptoms.—The disease usually begins with congestion of the nasal mucous membrane and the development of a pea-sized, circumscribed swelling on the nasal septum. The patient soon complains of malaise, headache, severe neuromuscular pains in the neck and shoulders, constipation, and fever. The fever is moderate in degree and usually subsides in from three to five days, concurrently with subsidence of the nasal symptoms. The swelling of the nasal mucous membrane never goes on to suppuration, but cases have been reported in which sudden subsidence of the swelling has been followed by grave cerebral symptoms, such as coma, delirium, and death. The spleen is usually enlarged and palpable. A diffuse erythematous eruption has occasionally been observed.

Treatment.—The treatment is purely symptomatic. Puncture of the spleen, which has been recommended as affording relief to the local and general symptoms, seems to be unduly hazardous.

JAPANESE RIVER FEVER.

Japanese river fever is an acute, infectious disease, occurring in the western part of the Island of Nippon, and characterized by an initial eschar, soon followed by ulceration of the skin, continued fever, inflammation of the lymphatics, and an exanthematous eruption.

Etiology.—The nature of the disease is unknown, though it affects practically only those engaged in harvesting the corn and hemp in a certain part of the island of Nippon. The Japanese attribute it to the bite of an ascarus. The primary eschar is not unlikely the point of infection. Sex and age are without etiological influence. One attack does not confer immunity, though it seems to lessen the severity of subsequent attacks.

Symptoms.—The period of incubation is from four to seven days, during which the patient may complain of malaise and repeated rigors.

The first indication of the disease is the initial eschar, which develops about the genitals, the axillæ, or the neck. Soon an ulcer forms—round, dark, tough, firmly adherent, and surrounded by a painless area of superficial congestion. The regional lymph nodes, and sometimes all the lymph nodes of the body and the spleen, become swollen and congested. With the onset of the infection, fever develops, pursues a more or less continued course, and ranges between 103° and 104° F. Catarrhal inflammation of the conjunctivæ and of the bronchi not infrequently arises. On about the fifth to the seventh day an eruption, consisting of large, dark red papules, appears on the face, forearms, legs, and trunk, and not infrequently at the same time a minute lichenous rash appears on the forearms and trunk. Both eruptions disappear in from four to seven days, while the fever begins to remit at the end of about two weeks, and the temperature soon reaches the normal. During the second week the ulceration begins to heal, the eschar being first cast off. The mortality varies between 15 and 50 per cent.

Treatment.—The treatment is purely symptomatic. The ulcer should be dressed antiseptically. Quinine and the salicylates have been recommended internally. Hydrotherapy also may be useful.

PONOS.

Ponos is a rare, chronic, endemic disease of childhood that occurs in the islands of Spetza and Hydra, off the coast of Argolis, and is characterized by irregular fever, enlargement of the spleen, and emaciation.

Etiology.—Apparently ponos effects only children, and commonly those under the age of four years, and of tuberculous antecedents. It appears to be uninfluenced by climate, season, or social conditions. Its exciting cause is not known, and in reality its nature is ill understood.

Pathology.—Apparently the necrospy findings of only one case have been reported. In this the spleen was much enlarged, the capsule firm, and the stroma and trabeculæ considerably thickened; the liver exhibited early cirrhosis; the kidneys were congested; and the bronchial and the mesenteric lymph nodes were swollen.

Symptoms.—The onset is sudden with fever, which, having developed, pursues a remittent and irregular course. The spleen rapidly enlarges and becomes tender; dyspepsia and constipation develop, and the patient emaciates rather rapidly. When the disease has become well established œdema and ascites may develop, and hemorrhages into and from the different organs and tissues, especially the gums, may occur. Boils and superficial areas of gangrene have been observed. The disease lasts for a few months to several years, and the patients rarely recover.

Treatment.—The treatment is unsatisfactory and scarcely more than symptomatic, consisting largely of careful dieting and the administration of tonics.

HILL DIARRHŒA.

Hill diarrhœa is a peculiar disorder of the tropics, especially India, characterized by morning diarrhœa, copious, pale, liquid stools, and flatulent dyspepsia.

Etiology.—The causative factors of hill diarrhœa are ill understood. The disorder is especially common in colonists who, having become acclimated to low, hot plains, go (often for a holiday) to the neighboring hills (5000 feet or more). The disease seems to be more than an ordinary intestinal catarrh; in some respects it resembles sprue, though, unlike it, it tends to recovery. By some observers it is believed to be dyspeptic in character and associated with suppression of the function of the liver, pancreas, etc. Scheube attributes it to atony of the colon, and to chilling of the abdomen due to the cold and dampness of the early morning hours of tropical mountainous countries.

Symptoms.—The disorder not infrequently begins with flatulent dyspepsia soon after the subject reaches the mountains. Usually on the following morning between 3 and 5 o'clock diarrhœa begins, and from six to twelve stools or more may be passed before noon, after which the diarrhœa not infrequently ceases for the remainder of the day. The stools are copious, watery or pasty, pale, frothy, and have been likened to whitewash (they contain no biliary coloring matter). The stools are, as a rule, unattended by pain or tenesmus, though there may be a vague, indefinite tenderness along the course of the colon. The abdomen is sometimes markedly distended, and there may be considerable borborygmi. The disease tends to recovery at the end of ten days or two weeks. Recurrences may attend repeated visits to the hills.

Diagnosis.—Manson states that the distinctive features of the disease are the regular recurrence of morning diarrhœa, its cessation after a certain hour in the forenoon, the absence of color in the stools, and the attendant flatulence.

Treatment.—The diet in the majority of cases should consist of milk, though one can readily understand that some patients might do better on animal broths, etc., that have less tendency to ferment. The patient should remain in bed during the morning hours, and should be warmly clad. Calomel is believed to be useful to stimulate the hepatic function. Crombie and Manson advise a teaspoonful of Liquor hydrargyri perchloridi (B. P.), in water, fifteen minutes after each meal. The digestive ferments are sometimes useful. If treatment is of no avail, the patient should return to the lowlands whence he came.

SPRUE.

(*Psilosis; Chronic Diarrhœa of Warm Climates; Chronic or Endemic Diarrhœa of Cochin China; Tropical Diarrhœa; Ceylon Sore Mouth; Cachexia Aphthosa.*)

"Sprue, or psilosis, may be defined as an insidious, chronic, remitting inflammation of the whole or part of the mucous membrane of the alimentary canal, occurring principally in Europeans who are residing or have resided in tropical or subtropical climates. It is characterized by irregularly alternating periods of exacerbation and comparative quiescence; a peculiar inflamed, superficially ulcerated, exceedingly sensitive condition of the mucous membrane of the tongue and mouth; great wasting and anemia; pale, copious, and often loose, frequent, and frothy fermenting stools; very generally by more or less diarrhœa; and also by a marked tendency to relapse. Sprue may either be primary, or it may supervene on or complicate other affections of the alimentary canal. Unless properly treated it is usually fatal." (Manson.)

Etiology.—The cause is unknown. The anatomical lesions are not characteristic.

Treatment.—The treatment is largely hygienic and dietetic. Manson prefers an exclusive milk diet for weeks or months; others prefer a meat diet.

EPIDEMIC GANGRENOUS PROCTITIS.

Epidemic gangrenous proctitis is an acute infectious disease, characterized by rapidly spreading phagedena of the anus and rectum, progressing to prolapse and gangrene of the ulcerated rectum.

Etiology.—Epidemic gangrenous proctitis is more or less widely distributed throughout the tropics, but it is especially prevalent in Central and South America, and in the Philippines and other Pacific Islands. Its exciting cause is not definitely known, but it has been attributed to bacterial infection (Bacillus dysenteriæ, Bacillus pyocyaneus, etc.), and in Venezuela to the eating of the green stalks of unripe maize. The latter view, however, is scarcely tenable (for all cases at least), since the disease occurs where maize does not exist. The development of the disease seems favored in low-lying, damp regions. In South America it is almost confined to children; in other tropical regions adults also are attacked.

Pathology.—The lesions consist of ulceration attended by pseudomembranous formation, limited in some cases to the region of the sphincters, but often spreading to the rectum and the sigmoid, and soon followed by prolapse and gangrene of the rectum.

Symptoms.—The initial symptoms consist of burning and itching above the anus and a tendency to frequent defecation. Soon the discharges become more frequent and are attended by abdominal pain and constant and distressing tenesmus, which frequently leads to extrusion

of the bowel. Fever, anorexia, and thirst supervene. The stools at first consist of fecal matter; soon, however, they become mucoid or muco-sanguineous, and extremely fetid. Occasionally even almost pure blood is discharged. Severe nervous symptoms, such as delirium and coma, and in children, convulsions, supervene. The patient emaciates rapidly, and, as a rule, soon dies—in some cases before prolapse of the rectum has occurred. Recovery, however, occasionally ensues, even after the advent of coma, prolapse, and gangrene (with sloughing) of the rectum.

Diagnosis.—In the early stages the disease is not unlikely to be mistaken for dysentery, but an examination of the anus and rectum serves to prevent error.

Treatment.—Opium is indicated to relieve pain and to control the intestinal movements. It may be given hypodermicly, but should also be applied locally to the inflamed and ulcerated areas. Antiseptic and soothing lotions to the anus and rectum are also called for. The local use of a portion of roasted lemon or of lemon juice has a vogue in Venezuela. In the event of gangrene the rectum must be excised, should the patient's condition warrant it.

TROPICAL PHAGEDENA.

Tropical phagedena is a rapidly spreading gangrene of the skin and subcutaneous tissues, leading to the formation of a large sloughing sore.

Etiology.—Tropical phagedena is preëminently a disease of the tropics, occurring especially where the climate is hot and damp. The exciting cause of the disease is not known, though doubtless it is a microörganism. It finds its readiest victims among the physically depressed and those in poor health from any cause. In some subjects an insect bite or an abrasion seems to be the starting point of the infection.

Symptoms.—The feet and legs are the most common seat of the lesions, though no part of the body is exempt. The first indication of the disease, if the skin has been previously sound, is the development of a large blister, containing a serosanguinolent fluid. This ruptures in a few hours, and exposes a sloughing sore which rapidly spreads and forms a horribly fetid, yellowish ulcer, the centre of which liquefies in a few days, while the periphery continues to spread. Not only is there peripheral extension, but also extension in the depths, whereby the muscles, tendons, nerves, vessels, and bone may become implicated. At the end of a week or more the slough is gradually separated. When the disease begins in an already existing wound or sore, the granulating surface dries and is gradually converted into a slough.

The lesions cause considerable pain which, together with the drain on the system, undermine the patient's health, and may lead to death from exhaustion, hemorrhage, or septicemia. In some cases, however, the constitutional disturbance is relatively slight and cicatrization proceeds —provoking, as a rule, considerable deformity and mutilation.

Treatment.—Opium is indicated not only to relieve pain, but also because of a seeming beneficial action on the phagedenic process. The ulcer should be curetted or cauterized with pure carbolic acid, and then continuously irrigated with a tepid antiseptic lotion. The general health should be maintained by tonics and nutritious food.

TROPICAL BUBO.

(*Climatic Bubo; Tropical Non-venereal Adenitis.*)

Tropical bubo is a non-venereal inflammatory enlargement of the inguinal lymph nodes attended by remittent fever lasting from three to four weeks.

Etiology.—Tropical bubo is most common in the East Indies and China, but occurs also in South Africa, Japan, the Philippines, along the coast of the Mediterranean Sea, in the West Indies, Malaga, etc. Limited epidemics have been observed among sailors and soldiers. Age is of no etiological importance. The exciting cause of the disease has not been isolated, though it is doubtless microörganismal. Infection is believed to occur through small wounds or abrasions of the legs or genitals or through the bites of insects.

Symptoms.—The onset of the disease is often rather sudden, with chill, fever (103° to 104° F.), headache, backache, and enlargement, tenderness, and redness of the lymph nodes of the groin of one or both sides. In some cases a group of nodes, in other cases separate contiguous nodes, are involved. The nodes increase in size until they attain that of a goose egg, whereupon the fever, which has been remittent in type, gradually lessens and the nodes decrease in size—though they may remain enlarged for two months or more. Usually there is little pain, and the constitutional symptoms, aside from the fever are slight. In some cases, however, the skin over the enlarged nodes becomes adherent, and the nodes soften and form suppurating sinuses, which lead not only externally but often deeply into adjacent tissues. In this event the pain and constitutional symptoms may become marked. The suppuration may continue several months.

Treatment.—If softening has not occurred, iodine and ichthyol, or an ointment of mercury, belladonna, and iodine, may be applied. If softening occurs, the abscess should be opened freely, curetted, and treated antiseptically. General treatment is rarely called for, and at all events is to be based upon general principles.

ULCERATING GRANULOMA OF THE GENITALS.

Ulcerating granuloma of the genitals (pudenda) is a chronic venereal disease of the tropics, characterized by its granular character, its large extent, and its tendency to hemorrhage.

Etiology.—Ulcerating granuloma of the genitals occurs in men and in women between the ages of thirteen and fifty years, and is uninfluenced by sex. The exciting cause has not been isolated, though it is present in the secretions from the granulations which are auto-inoculable and infectious to others. Wise has demonstrated spirochetes (resembling Spirochæta refringens) in the secretions.

Symptoms.—The disease begins on the genitals as an insignificant circumscribed nodular thickening and elevation of the skin, which soon develops into a light red, shiny mass of granulation tissue (partaking of the nature of the infectious granulomas); this has a fetid odor, excretes a thin, sanguineous fluid, and bleeds readily. The warm, moist surfaces about the scrotum, or the labia, and the thighs, are most susceptible of infection. The lesions spread slowly by peripheral extension, as well as by auto-inoculation of opposed (and other) surfaces, and involve most frequently the scrotum, the perineum, the glans penis, and the upper part of the thighs in the male, and the labia, the vagina, and the upper part of the thighs in the female. Spontaneous recovery is unusual, though not infrequently attempts at healing are seen—gradual shrinkage and the formation of a thin, somewhat raised, cicatrix, which, however, having formed, not uncommonly breaks down again.

Treatment.—Complete excision is the best treatment if the lesions are not too extensive—in which event the granulation tissue should be curetted and cauterized.

VERRUGA.

Verruga is a chronic infectious disease, characterized by initial (so-called) rheumatoid pains, fever, anemia, and the development of peculiar, warty, granulomatous, protuberances on the skin, mucous membranes, and the internal organs.

Etiology.—Verruga is most prevalent in Peru (whence it is often called verruga peruana), on the Pacific slope of the Andes Mountains, and in certain of the mountainous regions of Chili, Bolivia, and Ecuador. It occurs in both sexes, at all ages, and is apparently favored by moisture, heat, and elevation (since it is scarcely if ever observed at the level of the sea). It occurs in circumscribed epidemics, but is not markedly contagious. The exciting cause has not been isolated. How infection is spread is not known, though the natives of Peru attribute it to the water of certain springs. Malaria is frequently associated, and is believed by some to be of etiological significance. One attack confers immunity.

Symptoms.—The period of incubation is usually from fifteen to forty days, though rarely it may be longer or even shorter. Clinically two well-defined stages are recognized—that of invasion and that of eruption. The onset is usually insidious and marked by lassitude, indisposition, neuromuscular pains in the legs, followed in a few days by slight rigors and evening fever, which soon increase in severity. The

fever may reach 102° to 103° F., and pursues usually a remittent, but occasionally an intermittent course. The fever is accompanied by insomnia, intense headache, hemorrhages, and pains in the back and the joints, of which the knee, ankle, and the small joints of the hands and feet are most susceptible.

Spasmodic and painful contractures of certain muscles, especially of the calves and of the neck, occur—most frequently at night. Nausea, vomiting, vertigo, photophobia, transient amblyopia, dyspnœa, bronchitis, loss of appetite, and marked thirst are also commonly complained of. Early in the disease the bowels are constipated, but later diarrhœa develops. Marked anemia and emaciation soon supervene, and constitute characteristic features of the disease. The liver is frequently, the spleen less commonly, enlarged and palpable. The fever gradually subsides in about thirty days (more or less) and therewith the stage of eruption sets in. This consists at first of pin-sized pinkish papules, which gradually become darker (somewhat bluish) in color, and increase in size until they form quite large excrescences. Manson originally regarded them as identical macroscopically and microscopically with the granulomas of yaws, but more recently other observers, notably Glogner, have pointed out noteworthy differences. As in yaws, however, the eruption may be sparse or abundant, discrete or confluent; and the swellings or granulomas are exceedingly vascular and bleed freely, thus contributing to the anemia. They are found most commonly on the face, neck, and limbs; in many cases they develop on the palms of the hands, the soles of the feet, and the hairy scalp—while the trunk is usually exempt; they are common also on the mucous membranes, and in the different organs of the body, where they often occasion notable hemorrhages. The lesions persist from four to six weeks, and then desiccate and desquamate, or sometimes undergo ulceration and suppuration.

Prognosis.—Among natives the mortality is from 10 to 15 per cent.; it is much higher among the white race, and may reach 60 to 70 per cent.

Diagnosis.—Though verruga much resembles yaws, it can usually be distinguished by the involvement of the internal organs, which are unaffected in yaws, and by the marked tendency to hemorrhage manifested by the lesions.

Treatment.—Treatment, aside from removal from mountainous regions to the coast, which is indicated in all cases, is purely symptomatic. Quinine has been recommended on account of the common association of malaria. The removal of the ulcerated areas has also been practised.

KUBISAGARI.

(*Endemic Paralytic Vertigo.*)

Kubisagari is a chronic endemic disease of the northeastern part of the principal island of Japan, characterized by dimness of vision,

diplopia, ptosis, and paralysis of certain muscles, especially of the neck (whence the name, kubisagari, that is, one who hangs his head).

Etiology.—The nature of the disease is ill understood. It is almost confined to farming peasants, especially those who live under the same roof with their cattle, in which, as well as in many other respects, it resembles endemic paralytic vertigo (Gerlier's disease) of Switzerland and certain parts of France. Miura believes that the local horse plague and cattle plague are in some way related etiologically to the disease. Age and sex are without influence. The disease is most common during the warm season, and disappears during the winter.

Symptoms.—Kubisagari is characterized by attacks which come on at intervals of several hours to several days, and last for from several minutes to several hours, during which there is dimness of vision, diplopia, ptosis, and paralysis of different muscles. Paralysis of the posterior cervical muscles is the most common, and causes the head to drop forward; paralysis of the muscles of the tongue, of the lips, of the cheek, and of deglutition interferes with speech, mastication, and swallowing; and paralysis of the muscles of the trunk and the extremities, which is less common, interferes with station and locomotion. The attacks sometimes come on without known cause, but in some cases are induced by physical exertion (especially, it is said, a stooped posture with the stomach empty), by hunger, by indigestion, by eye-strain, etc. In some cases there is considerable mental depression; the lacrymal, nasal, and salivary secretions may be increased; and the reflexes may be exaggerated. The disease is not fatal, though it may last for years.

Treatment.—Treatment, as a rule, is not encouraging. Miura reports favorable results from the use of potassium iodide and arsenic, and the bromides are believed to be sometimes of some avail in controlling the frequency of the attacks.

GANGOSA.

Gangosa is a disease that prevails in Guam, where it is said to involve about two per cent. of the population; it occurs also in the Ithmus of Panama, the West Indies, the Philippines, and perhaps in certain parts of South America. It is described by Mink and McLean as being characterized by a destructive ulceration, usually beginning on the soft palate, pillars, or uvula, and extending by continuity to the hard palate, the nasal cavity, the larynx, and even the face. Active ulceration is followed, after a variable period, by cicatrization or chronic ulceration. Mutilation always results. Constitutional symptoms are either slight or absent. The disease resembles, but may readily be differentiated from, leprosy, syphilis, tuberculosis, etc. A specific infectious agent has not been isolated; but the disorder appears to be infectious, being spread by contact.

AINHUM.

Ainhum is described by Manson as a disease affecting the toes, especially the little toes, of negroes, East Indians, and other dark-skinned races, and commencing as a narrow groove in the skin, almost invariably on the inner and plantar side of the foot of the little toe, of one or both feet. Once started, the groove deepens and extends gradually around the whole circumference of the toe; the distal portion of the toe is likely to swell to a considerable size, as if constricted by a ligature. There is little or no pain, although there may be inconvenience from the liability to injury of the dangling and everted toe. In the course of years the groove deepens, and finally the toe drops off as if amputated. The disease has been variously interpreted—as being a manifestation of leprosy, a circumscribed scleroderma, a trophoneurosis, a congenital spontaneous amputation, the result of self-mutilation by ligatures, wearing toe rings, etc., and as caused by injuries to the toes. Wellman believes that it results from injuries to bare-footed persons, caused by long grasses, and that the factor in prolonging the irritation and inflammation is the chigger, Sarcopsylla penetrans, which seems to have a predilection for the affected spot, especially if the skin be cut or torn.

GOUNDOU.

(*Anakhre.*)

Goundou is a disease that occurs among the natives of Africa, and is characterized by severe and persistent headache, later followed by a sanguinopurulent discharge from the nostrils, and the development of painless symmetrical swellings at the sides of the nose. Although the discharge may subside, the swellings progressively increase in size until they may be as large as an ostrich's egg; eventually they may obstruct the line of vision or destroy the eyes. The disease has been variously interpreted as a manifestation of yaws, or of syphilis, as a malformation due to the non-union of the nasal and frontal bones, as a manifestation of atavism referable to tribal peculiarities of the original negro, as a disease *sui generis*, and as a disease caused by the larvæ of insects in the nostrils. Wellman believes that the growths are due to an osteoplastic periostitis, the definite cause of which has not been demonstrated.

PSITTACOSIS.

Psittacosis is a disease of birds, characterized by loss of appetite, general weakness, diarrhœa, and death (often in convulsions). It is believed to be communicable to man, infection being commonly acquired from parrots—in England, Germany, France, and Italy. In man it is characterized by pneumonia that is often atypical in course, attended by toxemia, and usually ends fatally.

SECTION II.

INTOXICATIONS.

The intoxications to be here discussed comprise the series of diseased conditions, the result of more or less continued activity of divers poisonous chemical substances; the acute poisonings belong more properly to the subject of toxicology.

ALCOHOLISM.

Symptoms.—Acute Alcoholism.—The effect of a large amount of alcohol taken within a short time is exerted chiefly on the nervous system; at first there is exaltation of the nervous functions, which is soon followed by perversion, and finally depression and stupor. In the ordinary case the action of the heart becomes at first accelerated, respiration quickened, the muscular force and activity increased, the mentality active, the eyes brilliant, and the face flushed (although it may be pale); the patient gradually becomes loquacious and indiscreet in his confidences, and care fades away. Soon, however, the perceptive faculties become confused, the intellect dulled, the memory faulty, movement of the limbs uncertain and incoördinate, the gait unsteady, the speech thickened, the facial flush deepened, the pupils dilated, the conjunctivæ red and watery, and the gaze staring. Gradually depression of the general functional activities ensues, and the patient passes into a more or less profound sleep (from which, however, he can be more or less aroused); the temperature may become subnormal. In other cases the subject becomes readily infuriated or maniacal, and upon slight or no provocation may commit serious crimes, such as murder, etc. Periodical recurrences of such attacks of acute alcoholism are common in persons with an inherited or acquired craving for alcohol—so-called dipsomaniacs; in the intervals between attacks the subject may abstain entirely from alcohol. The ordinary attack of acute alcoholism is readily recognized, but alcoholic coma must be distinguished from apoplexy, uremia, diabetic coma, morphine poisoning, etc.

Chronic Alcoholism.—While it is undoubtedly true that many persons can take a moderate amount of alcohol (whiskey, wine, beer) and attain a ripe old age in good physical condition, the matter is

largely individual, and to the majority of mankind the continuous or daily consumptiom of even a moderate amount of alcohol is fraught with danger. Alcohol brings inevitably in its wake disordered function and altered structure. The bloodvessels become sclerosed, and the heart hypertrophied and dilated or fatty; the gastro-intestinal tract becomes congested and chronically inflamed, giving rise to the so-called morning sickness of alcoholics—anorexia, nausea, perhaps vomiting, coated tongue, constipation, etc.; the liver becomes cirrhotic; the kidneys become the seat of chronic diffuse indurative nephritis; and the nervous system reveals various deteriorations. Of these, muscular tremor, involving the extremities, especially the hands, the lips, and the tongue, are common; various disorders of sensibility, including the special senses, neuralgiform pains and true neuritis, meningitis, etc., may develop. The mind (will and intellect) and the morals deteriorate. The patient becomes irritable and suspicious; his judgment becomes impaired, his memory enfeebled, and sleep unsatisfying; lapses from moral rectitude (sexual and other) are common; hallucinations and delusions may supervene, and the patient may gradually pass into irremediable insanity. The association in chronic alcoholics of loss of memory, pseudoreminiscences (false notions as to time, place, and actualities), and peripheral neuritis is spoken of as Korsakoff's psychosis.

Delirium Tremens (Mania à potu).—Delirium tremens is an incident of chronic alcoholism, that usually follows periodical excessive indulgence in alcohol, and may be precipitated by a fright, shock, fracture, or other trauma, acute infection, especially pneumonia, etc. The symptoms usually begin with general restlessness, mental activity (sometimes apathy), and insomnia—for which additional alcohol may be taken—without avail. In two or three days delirium and hallucinations of sight and hearing set in. The patient talks incessantly and incoherently; conducts imaginary enterprises, and insists upon their urgency, so that unless watched or restrained he avails himself of the first opportunity to escape from his bed and room—usually by way of the window. Should he doze, he may be awakened by terrifying dreams, and while awake he is tormented by the most distressing hallucinations —of sight, such as rats, mice, demons, snakes (a patient of mine with marked aortic insufficiency was assured of being infected with snakes by watching the violent pulsations of his very tortuous arteries in the arms); and of hearing, insulting remarks, threats of imaginary enemies, etc. There is a well-marked muscular tremor; the pulse is rapid and soft, often very weak; and there may be some fever, which, however, is almost if not always due to a complication, such as pneumonia, which is often overlooked. In favorable cases, at the end of three to five days the patient sleeps, the restlessness abates, the hallucinations disappear, and the patient begins to convalesce. Very noticeable improvement often follows a prolonged sleep, but the tremor may continue for some time. In unfavorable cases with increase of the symptoms and persisting insomnia the patient dies of exhaustion.

Treatment.—The ordinary and recurring attacks of acute alcoholism rarely require any treatment per se. The effects of the alcohol pass off during the sleep, and although the patient shows for some days the grind of the mill of the gods, he soon recovers. Should, however, the coma be deep, gastric lavage should be employed. If the patient is unruly or maniacal, a hypodermic injection of apomorphine ($\frac{1}{6}$ grain, 0.01 gram) usually induces not only nausea and vomiting, but also quietude. In the event of convulsions, a few whiffs of chloroform may be given, or chloral (20 grains, 1.3 gram) and bromide (40 grains, 2.6 gram) may be administered, and repeated if necessary in two hours. If the patient is much depressed (low temperature, etc.), resort should be had to energetic frictions, external heat, enemas of hot saline solution and coffee, and coffee by the mouth (or stomach tube), hypodermic injections of strychnine and atropine, artificial respiration, the faradic current to the respiratory muscles, etc.

The chronic alcohol habit is best treated in a hospital or similar institution where the physician has entire command of the situation. The alcohol should be withdrawn as rapidly as possible (three to five days); there is neither logic nor advantage in continuing it in gradually decreasing amounts for two or three weeks. The following may be given as a substitute for the alcohol:

℞—Tincture of nux vomica	6 drams	25	0
Tincture of capsicum	2 drams	8	0
Ammonium bromide	5 drams	20	0
Elixir of ammonium valerianate	2 ounces	60	0
Syrup of ginger	2 ounces	60	0
Water, sufficient to make	8 ounces	250	0—M.

S.—One tablespoonful (15 c.c.) four times a day.

Atropine hypodermicly, $\frac{1}{120}$ grain (0.0005 gram), twice or thrice daily, will keep in control the nervousness in the early days of abstinence from the alcohol. An additional dose of a bromide, or chloral (20 grains 1.3 gram), or hyoscine ($\frac{1}{100}$ grain, 0.00065 gram), or sulphonal or veronal, etc., may be given at bedtime to induce sleep. Good results also attend frequent feedings, perhaps of highly seasoned food, for a time at least (to satisfy the craving for something stimulating); free catharsis; a daily or twice daily hot bath followed by a cold douche; and some outdoor exercise.

If the alcoholic subject is seen in delirium tremens or at the end of a debauch ("threatened with delirium tremens"), the first essential is to get him asleep; often he will awake from a prolonged sleep entirely refreshed, and the danger has past. He must be kept in bed (preferably on a mattress on the floor) and gently restrained; the use of wrist- and ankle-straps may be necessary, but forcible personal restraint by nurses or attendants increases the delirium and resistance. The patient should first be given 5 grains (0.3 gram) of calomel and 10 grains (0.6 gram) of compound jalap powder dry on the tongue, and then a hypnotic —chloral (15 grains, 1 gram) and morphine ($\frac{1}{8}$ grain, 0.008 gram) are the best; these doses may be repeated at the end of an hour if the patient

is still wakeful and restless, but in the event of a weak heart this should be fortified with strychnine and digitalis. To the chloral and morphine one may add sodium bromide (40 to 60 grains, 2.6 to 4 grams); or he may give hyoscine ($\frac{1}{100}$ grain, 0.00065 gram), paraldehyde (1 to 2 drams, 4 to 8 grams), etc. A. Lambert advises:

℞—Strychnine sulphate	$\frac{1}{30}$ grain	0.002
Hyoscine sulphate	$\frac{1}{100}$ grain	0.00065
Apomorphine hydrochloride	$\frac{1}{10}$ grain	0.006

The use of atropine ($\frac{1}{100}$ to $\frac{1}{60}$ grain, 0.00065 to 0.001 gram) twice daily has also been attended by good results.

Hypnotics, such as trional, veronal, and the bromides, usually suffice after the first prolonged sleep, but none may be needed. Special attention must be given to supporting the patient's strength by the use of strychnine ($\frac{1}{30}$ grain, 0.002 gram, every four hours) or nux vomica, and by an abundance of nutritious food—preferably liquid and hot (milk, broths, eggs), perhaps highly seasoned, for several days. A weak heart must be supported on well-known principles. Lambert extols the use of the belladonna, xanthoxylum (prickly ash), and hyoscyamus mixture mentioned under the opium habit.

OPIUM HABIT.

(*Morphine Habit; Morphinism; Morphinomonia.*)

Taken, as a rule, in the beginning to alleviate pain or to produce sleep, or, as in the East, for reasons similar to those that actuate the Occidental to take alcohol, opium or morphine, and to a less extent codeine and heroin, gradually create an almost if not quite uncontrollable craving. The habitues, both the pleasure seekers (morphinists) and the pain-racked subjects (morphinomaniacs) may take enormous amounts—a half to one ounce of opium, a pint of tincture of opium, 10 to 30 grains of morphine, etc., daily, and the amount is usually increased with the passage of time.

Symptoms.—The opium (or morphine) habitue usually has a pale, grayish-ashen countenance, and deep-sunk, dreamy, inexpressive eyes; he (or she) is withered, emaciated, and prematurely old-looking, restless, and irritable. When not under the influence of the drug the patient is likely to be depressed, weak, and inactive. As a rule, the appetite is impaired and capricious, digestion deranged, the bowels constipated, and sleep poor and unrefreshing. Itching of the skin is not uncommon, and there may be œdema, etc. Marked perversion of the moral sense is common; the habitué has no regard for his word, either in statement of fact or promises. Neurasthenic and hysterical phenomena are common in women. Extraordinary suffering and collapse may follow sudden discontinuance of the drug.

Treatment.—It is worse than useless to attempt treatment outside of a hospital or sanatorium where the strictest supervision of the patient

is possible; the ordinary morphine habitue usually goes to a hospital laden with morphine, and will obtain the drug at all hazards. As a rule, the patient should remain in bed for ten days or two weeks. The morphine should be withdrawn rather rapidly, that is, within a week. At the same time the patient should receive a cold tub bath or sponge bath in the morning, and a hot bath at night; and massage in the middle of the day, or in the afternoon. An abundance of simple nutritious diet should be given at short intervals. Much of the abdominal distress, and pain, nausea and vomiting, diarrhœa, etc., has been attributed to hyperchlorhydria, which may be met by the aforementioned frequent feedings, large doses of sodium bicarbonate, etc. Restlessness and nervousness may be controlled with the bromides, valerian, etc.; sleep may be promoted by an occasional dose of sulphonal, veronal, chloral, etc., if required, but caution is necessary lest one drug habit be substituted for the other. The cardiac and general asthenia may require the use of strychnine, aromatic spirit of ammonia, and other diffusible stimulants, strophanthus, digitalis, etc. Lambert extols the following as a specific in the treatment of drug (morphine, cocaine, alcohol) addictions:

℞—Tincture of belladonna	2 ounces	60 0
Fluid extract of xanthoxylum,		
Fluid extract of hyoscyamus	of each 1 ounce	30 0

Lambert advises that the treatment be begun with 4 compound cathartic pills and 5 grains of blue mass, and a soapsuds enema. When the pills have begun to act, 6 to 8 minims of the belladonna mixture should be given every hour, and every six hours the dose should be increased 2 minims, until 14 to 16 minims is being taken hourly, or the patient has shown signs of belladonna intoxication (dilated pupils, dryness of the throat, a red rash, rapidity and incisiveness of speech, or beginning delirium). Should these develop, they should be allowed to subside, whereupon one should begin with the mixture again in 8 minim doses hourly. With the first dose of the mixture, from one-half to two-thirds of the total daily amount of morphine or cocaine to which the patient has become accustomed, is given—in three equal doses at half-hour intervals. The bowels must be opened thoroughly—with compound cathartic pills, blue mass, calomel, castor oil, etc. At the end of thirty hours strychnine, digitalis, or strophanthus should be commenced. The treatment is said to take away the desire for morphine, cocaine, or alcohol in less than five days, with a minimum of discomfort and suffering to the patient.

COCAINE HABIT.

(*Cocainism.*)

The cocaine habit is usually acquired in consequence of taking the drug to relieve exhaustion (sexual, postalcoholic, etc.), or to alleviate

pain, neuralgia, chronic nasal catarrh, etc. As much as 30 or 40 grains daily may be taken by the mouth or hypodermicly.

Symptoms.—Cocaine at first produces a feeling of mental exhilaration and bodily vigor, freedom from pain and distress, and a subjective sense of the ability to accomplish the mind's desires and the heart's cravings; but the judgment becomes perverted and accomplishment by no means keeps pace with stimulated imagination. In the course of time these feelings give way to nervousness, irritability, introspection, apprehension, and insomnia. As the use of the drug is persisted in, the periods of peace and exhilaration become shorter, and the restlessness, depression, and distorted mentality more marked; the appetite fails, the patient emaciates, and even insanity may ensue. Some subjects exhibit a periodicity in their addiction to the drug; and others are addicted also to alcohol, morphine, etc., concurrently or intermittently.

Treatment.—The treatment in general should follow the principles suggested under morphinism. Withdrawal of the cocaine should be moderately rapid—within a week, the bromides, valerian, hyoscyamus being used to control the nervousness; or one may have recourse to the belladonna mixture advised by A. Lambert. Care should be taken not to substitute the alcohol, morphine, or chloral habit for the cocaine addiction.

LEAD POISONING.

(*Plumbism; Saturnisim.*)

Lead poisoning is a common result of one of the so-called dangerous industrial trades—lead working. Under like conditions women seem to be more susceptible than men. The lead usually enters the system by way of the respiratory tract (inhalation of lead-dust-laden atmosphere), by way of the digestive system (contaminated food and drink; professional administration of lead), or by way of the skin (cosmetics, hair dyes, thread, etc.). Less commonly poisoning may follow the use of lead douches in the vagina, etc.

Pathology.—In acute and fatal cases the conspicuous lesion is catarrhal gastro-enteritis and softening of the brain. Goadby and Goodbody believe that the essential and primary action of lead intoxication is the production of minute and microscopic hemorrhages in various portions of the body, including the nervous system, and that the clinical symptoms of lead palsy and its good prognosis when treated early are explainable by the presence of minute hemorrhages in the peripheral nerves. In advanced cases the lesions are those of multiple neuritis, with atrophy and degeneration of the muscles; degenerative lesions may also be found in the ganglion cells of the anterior cornua of the spinal cord.

Symptoms.—Acute Lead Poisoning.—Symptoms may develop after a few weeks exposure, or, rarely, they may not come on until after years of exposure. The manifestations are usually those of severe gastro-intestinal catarrh (nausea, vomiting, colicky pain, diarrhœa or con-

stipation), which may lead to collapse and death. There may be associated coma or convulsions, cerebral or peripheral paralyses, rapidly developing anemia, suppression of urine, albuminuria, slow pulse, etc.

CHRONIC LEAD POISONING.—The significant symptoms of chronic lead poisoning consist of lead colic, lead paralysis, lead encephalopathy, and lead cachexia.

Lead colic (*painter's colic*) is the most common manifestation. The pain, which may be preceded by ill-defined gastro-intestinal disorder, is usually very severe, paroxysmal (colicky), and is often diffuse, although it may be localized to the region of the umbilicus, the epigastrium, or hypogastrium; it is usually relieved by firm pressure. The paroxysms may continue for a few moments to an hour or more, and when the severe pain subsides more or less dull aching pain continues. Constipation is the rule, but the paroxysms of pain may be associated with diarrhœa. Relapses are common, and exposure may cause chronic lead colic.

Lead paralyses (or palsies) are quite common, and may come on suddenly or slowly. They are due to peripheral neuritis, and are associated with tremors, wasting of the muscles, reactions of degeneration, paresthesias, neuralgias, arthralgia, etc. The following types have been distinguished: (1) The musculospiral type (antibrachial type, wrist-drop), in which involvement of the musculospiral nerve leads to paralysis of the extensor muscles of the wrist and the fingers (except the supinator longus) and the characteristic wrist-drop. In long-continued cases slight backward displacement of the corpus and distention of the synovial bursæ (Grübler's tumor) may occur. (2) The brachial type, in which the deltoid, biceps, brachialis anticus, and sometimes also the pectoral muscles are involved. (3) The Aran-Duchenne type, common in tailors, in which the small muscles of the hand and the thenar and hypothenar eminences are involved (resembling chronic anterior poliomyelitis). (4) The peroneal type, in which the lateral peroneal and the extensor muscles of the toes are involved (steppage gait). Rarely there may be paralysis of the laryngeal or intercostal nerves; or a palsy resembling Landry's ascending paralysis may occur; or hemiplegia may ensue; or the condition may resemble tabes dorsalis.

Lead encephalopathy may be manifested by mild symptoms such as headache, dizziness, excessive irritability or depression, tremor, insomnia, dimness of vision, tinnitus aurium, etc., or by severe symptoms, such as delirium, coma, or convulsions. The delirium is marked by periods of violence and intervals of somnolence, which may be followed by true sleep and convalescence. Coma may come on suddenly or gradually and be preceded or attended by delirium or convulsions. The convulsion may be localized or generalized and resemble epilepsy.

Lead cachexia is characterized by a pallid, sallow complexion, coated tongue, a peculiar dry, metallic taste in the mouth, fetid breath, gastro-intestinal derangements, constipation perhaps alternating with attacks of diarrhœa, malaise, weakness, and emaciation.

Examination of the blood reveals a well-marked anemia—oligocythemia and oligochromemia (50 per cent. or thereabout), a characteristic basophilic degeneration of the erythrocytes, some erythroblasts, and perhaps slight leukocytosis. In most cases there is a bluish or grayish-black line (2 to 3 mm. wide) at the margin of the gums, in the papillæ (lead sulphide precipitated in the tissue by the sulphuretted hydrogen of the tartar); it must be distinguished from a somewhat similar-looking line between the gums and the teeth which is readily removed with a tooth-brush. The HCl content of the gastric juice is usually diminished or absent, and lactic acid may be present. Arteriosclerosis with hypertrophy of the heart and chronic interstitial nephritis eventually ensue.

Diagnosis.—The diagnosis is easy in the presence of any of the characteristic symptoms, but in a case of coma it may be exceedingly difficult. The bluish line in the gums and basophilic degeneration of the erythrocytes are of much importance. In other cases the first clue to the diagnosis may be a knowledge of the patient's occupation or of his exposure to the poison. In some cases lead may be detected in the urine.

Prognosis.—The outlook is favorable in all but the severe encephalopathies, which often result fatally, or if recovered from may eventuate in mental deterioration. Wrist-drop if neglected may prove intractable.

Treatment.—As soon as symptoms become in the slightest degree manifest, the patient should be removed from exposure to lead and warned against again subjecting himself to it, since there is always danger of relapse in susceptible individuals. Workers in lead should wear a mask, and should be scrupulously clean, especially in regard to the hands (finger nails), which often are the indirect source of intoxication (in eating, etc.). In the severe acute poisonings, sodium and potassium sulphate should be administered (to precipitate insoluble lead sulphate); opium should be given for pain, and stimulants for the collapse. Lead colic should be treated by magnesium sulphate, calomel and compound jalap powder; morphine and atropine hypodermicly for the pain and to relax the intestinal musculature; hot applications to the abdomen, etc. Potassium iodide (15 to 30 grains, 1 to 2 grams, daily) should be administered to promote elimination of the lead; iron should be given for the anemia, and massage, passive motion, exercises, and electricity for the local paralysis.

Carles has proposed the production of a sterile abscess (by the intramuscular injection of 1 c.c. of turpentine) with a view to withdraw into the abscess by means of the phagocytes the lead deposited in the body; and the later evacuating of the abscess.

ARSENICAL POISONING.

Arsenic may enter the system in poisonous amounts by the intestinal or the respiratory tracts. Most cases of poisoning are accidental, the poisoning being acquired in the pursuit of certain dangerous trades,

such as the manufacture of colored wallpapers, various fabrics, artificial flowers, etc., but poisoning may also follow the criminal administration or taking of Rough-on-rats, Paris green, etc.; occasionally it follows the therapeutic administration of arsenic.

Symptoms.—Beginning therapeutic overaction or intolerance of arsenic is manifest by salivation, epigastric pain and distress, nausea, vomiting, diarrhœa and tenesmus, œdema and itching of the eyelids, urticaria, pityriasis, psoriasis, eczema, muscular tremors, and contractions and stiffness of the joints. In what may be called acute poisoning the gastro-intestinal symptoms are aggravated, the urine and the feces may be bloody, and there may be anuria. In some cases gastro-intestinal symptoms are in abeyance and symptoms resembling opium narcosis may come on; later numbness, sharp darting pains, or paralyses may supervene. In chronic arsenical poisoning, the symptoms, which may develop insidiously, are usually those of increasing anemia and debility, pigmentation (also keratosis or herpes) of the skin, and multiple neuritis (paresthesia, neuralgia, paralyses), involving the legs (extensor and peroneal muscles) rather than the arms, and usually associated with rapid and marked atrophy of the muscles. There may be also divers gastro-intestinal disturbances, redness and bleeding of the gums, stiff joints, etc.; pseudotabes may ensue.

Diagnosis.—The diagnosis may be based upon known exposure to the influence of the poison, pigmentation and other lesions of the skin, multiple neuritis, and the detection of arsenic in the urine.

Treatment.—The treatment consists in avoidance of the source of intoxication; potassium iodide to promote elimination; iron for the anemia; and massage, electricity, etc., for the neuritis.

MERCURIAL POISONING.

(*Hydrargyrism; Mercurialism.*)

Mercury is extensively used in the manufacture of mirrors, bronzes, thermometers, decorative novelties, cosmetics, etc., so that poisoning not infrequently ensues. The poison may gain access to the system by way of the alimentary tract, the respiratory tract, or the skin. The therapeutic administration of mercury (calomel, blue mass, etc.) also may lead to poisoning.

Symptoms.—The acute symptoms consist of fetid breath, a metallic taste in the mouth, salivation, swelling, sponginess, and frequently hemorrhage of the gums, sometimes even loosening and shedding of the teeth, with ulceration and necrosis of the jaw. The characteristic of the chronic cases is muscular tremor (trembles), which at first occurs only on movement or excitement, but later becomes more or less continuous, usually first involving the tongue and face (interfering with speaking) and later the extremities (interfering with arm movements and walking). Physical strength lessens and mental deterioration may ensue.

Diagnosis.—The symptoms and the history are usually conclusive. Multiple sclerosis, paralysis agitans, and other disorders associated with tremors should be readily excluded.

Treatment.—The avoidance of the poison is the first essential of the treatment. Potassium iodide should be given to promote elimination. A gargle of tannic acid and antiseptics may be used during salivation. Iron, quinine, and strychnine in tonic doses should be given.

SNAKE-VENOM POISONING.

The common poisonous snakes of the United States are the Crotalus (rattlesnake), the Ancistrodon (water moccasin and copperhead), and the Elaps (coral and harlequin snake); in Europe only the vipers abound; in Asia, the cobra, vipers, crotalus, elaps, and other poisonous varieties are found. The venom is contained in a gland with a maxillary fang that becomes unsheathed in activity; the venom is viscid, and varies in color from pale amber to deep yellow. Noguchi states that snake venom owes its toxicity to the presence of several active principles, which are present in varying proportions in different venoms, whence the differences in their activity. These toxic principles are: That which produces instantaneous intravascular clotting of the blood; that which attacks the nervous system (neurotoxin); that which causes rupture of the walls of the capillary vessels and extensive hemorrhages (hemorrhagic principle); that which attacks the blood corpuscles, causing hemolysis and agglutination (hemolytic and hemo-agglutinative principles); those which cause lysis of other body cells (cytolytic principles); those which protect or harden the erythrocytes; and that which causes a loss of the bactericidal property of the blood.

Symptoms.—Noguchi states that the symptoms observed in man may be divided into local and general. The local symptoms consist in a rapidly appearing, inflammatory swelling at the site of the bite, followed by more or less ecchymosis and necrosis, with or without pain, and often with the appearance of lymphangitis and local phlegmon. The constitutional symptoms are fever, cerebral and spinal disturbances, especially of the medulla, including paralysis of the respiratory centre, and rapid advance of motor paralysis. Hematuria, hemoglobinuria, hematemesis, diarrhœa, vomiting, amaurosis, headache, vertigo, violent dyspnœa, and general convulsions are often observed. In viperine poisoning there may be also cold perspiration and a small intermittent pulse. Death may ensue in two minutes (Crotalus poisoning), more commonly after about fifteen minutes; or it may be delayed for from twelve to forty-eight hours, or longer.

Treatment.—An attempt should be made to retard the absorption of the venom, by the application of a ligature about the region of the bite, by opening the wound and cupping or sucking, or otherwise mechanically removing the venom. One should also inject into and

around the bitten region potassium permanganate (1 per cent.), calcium chloride (1.5 per cent.), chromic acid (1 per cent.), or ammonia. Alcohol, coffee, and tea may be used, as well as oxygen inhalations, and saline intravenous injections. Noguchi states that experimentally strychnine, morphine, nicotine, and curare are not only of no value, but distinctly harmful. The specific treatment is Calmette's antivenin, which should be used immediately if it is available. Unfortunately each venom seems to require for neutralization a specific antivenin; an efficient polyvalent antivenin has not yet been prepared.

FOOD POISONING.

(*Ptomaine Poisoning; Bromatotoxism.*)

Poisoning may follow the eating of food or pseudo-food: (1) Because the substance taken is in itself poisonous, that is, it contains an endogenous poison, and, speaking strictly, can not, therefore, be called a food (certain mushrooms, fish, etc.); or (2) because to an otherwise suitable food an exogenous poison may be added. In addition, some persons are unusually susceptible to the influence of, and become more or less ill from eating, certain foods, toward which mankind reacts in what may be called a standard manner, such as strawberries, buckwheat, veal, liver, etc. The exogenous poisons found in foods may be: (1) Various chemical adulterants added to preserve the food or to mask putrefactive processes, such as various sulphites, salicylic acid, boric acid, benzoic acid (and their salts), formaldehyde, potassium nitrate, etc.; or coloring matters, such as salts of copper, lead, arsenic, etc. (2) Metals, such as tin, lead, copper, etc., derived from vessels containing food. (3) Divers sorts of microörganisms—bacteria, fungi, and zoöparasites of specific infections, as well as the organisms of putrefaction. The alkaloidal products of putrefaction are spoken of as ptomaines, and their ill effects as ptomaine poisoning.

Meat Poisoning (Kreatoxism).—Meat poisoning may result from eating meat (especially sausage, ham, and pork, but also beef, veal, game, and all kinds of corned meat) infected with Bacillus botulinus (so-called botulism), Bacillus enteritidis (Gärtner), Proteus vulgaris, Bacillus coli, Bacillus paratyphosus, etc. The nature of the poison (kreatoxicon) has not been determined. The symptoms may be gastro-intestinal or nervous (toxemic), or both. Often there is a period of incubation of several hours or more, in which there may be no symptoms. The onset is often sudden, with abdominal pain, nausea, vomiting, diarrhœa, headache, weakness, and neuromuscular pains; there may be an initial chill or chilliness and subsequent fever (101° to 103°); headache, vertigo, faintness, and profuse sweating are common. In some severe cases the diarrhœa is profuse and quite exhausting (choleraic form), and with the toxemia may lead to collapse and death.

Milk Poisoning (Galactotoxism) and Cheese Poisoning (Tyrotoxism).—Poisoning may ensue from the use of milk and its derivatives (cheese,

ice-cream, etc.), the toxic agents being tyrotoxicon (Vaughan) and perhaps other ptomaines. In some cases the colon bacillus has been found in the milk. The symptoms consist of nausea, vomiting, diarrhœa, with perhaps bloody or cholera-like discharges, neuromuscular pains and cramps in the legs, fever, and collapse. There may be a rash resembling that provoked by belladonna.

Fish Poisoning (Ichthyotoxism) and Shellfish Poisoning (Mytilotoxism).—Fish poisoning may follow the eating of fish that has undergone putrefaction, or of certain parts (roe, testicles, poison glands connected with the fins, etc.) of otherwise wholesome fish. The symptoms may be gastro-intestinal (pain, nausea, vomiting, and diarrhœa), or nervous, with collapse, or they may resemble those of belladonna poisoning (dryness and constriction of the throat, dilatation of the pupils, ptosis, delirium, convulsions, coma). Shell-fish poisoning may follow the eating of unwholesome mussels, oysters, lobsters, crabs, etc. A poisonous principle (mytilotoxin) has been isolated in certain cases. The symptoms are largely gastro-intestinal (pain, nausea, vomiting, and diarrhœa), with which a rash and other symptoms suggesting belladonna poisoning are often added. The pulse may become rapid and the patient collapsed, or he may pass into coma.

Grain Poisoning (Sitotoxism).—Ergotism.—Ergotism results from eating rye and other grain infected with Claviceps purpurea (the ergot fungus). Kobert has isolated three poisonous substances—sphacelinic acid, cornutin, and ergotinin; to these Jacoby has added sphacelotoxin, which he believes to be the specific poison. The symptoms may be acute or chronic. The acute symptoms are largely gastro-intestinal with collapse. The chronic disorder may be of the gangrenous or the convulsive type. The gangrenous type is characterized by pain, paresthesias, anesthesias, blood stasis, and gangrene of the fingers, toes, nose, ears, etc. The convulsive type is characterized by a prodromal period of from ten to fourteen days, during which there may be pains and paresthesias, and perhaps fever; these are followed by muscular spasms and contractures, especially of the arms and legs, that may last for a few hours or days and recur. Delirium and convulsions may supervene. Eventually a pseudotabes (posterior spinal sclerosis) ensues.

Lathyrism (Lupinosis).—Lathyrism results from eating meal (and bread) to which vetch seed (Lathyrus sativus and Lathyrus cicera) has been added, usually as a substitute for wheat. It occurs in Italy, Austria, Africa, and India. The nature of the poison is not known. The chief symptoms consist of paresthesias, spastic paraphlegia (involving the legs), increased reflexes, etc.

Pellagra (Maidism).—Pellagra results from eating corn (maize) that has become in some unknown way unwholesome or spoiled. It is common in Italy, France, Spain and in the southern and western parts of the United States. The symptoms develop insidiously, the earliest manifestations usually being gastro-intestinal—anorexia, stomatitis, salivation, epigastric pain or distress, diarrhœa, and a gradually

increasing anemia, asthenia, disinclination to exertion, and psychic depression. The fully developed disease is characterized by cutaneous, digestive, and nervous symptoms. There is at first a characteristic pellagrous erythema that usually comes on first in the spring, tends to subside and recur (in the fall and spring). It develops bilaterally especially on the exposed surfaces, the hands, arms, face, and neck; that is, it seems to be related to the action of the actinic rays of the sun; it may be dry (usually early) or wet; the lesions become pigmented (liver-yellow or chocolate color) and usually progress to desquamation, exfoliation, and gangrene of the skin, which are followed by cicatrization. The characteristic digestive symptoms consist of stomatitis, the cardinal-red tongue (Watson), the bald tongue (Sandwith), or the stippled, bluish-black tongue (Lavender); salivation, pyrosis, and diarrhœa (fetid, slimy, greenish stools), sometimes bloody stools, may occur. The nervous symptoms consist of neuromuscluar pains in the back and legs, spinal tenderness, headache, vertigo, unilateral or bilateral mydriasis, muscular spasms, exaggerated reflexes, later paralysis with lessened or absent reflexes, mental depression, delusions, hallucinations, melancholia, and insanity. Mild cases may be afebrile, but fever (102° to 105° or more) is not uncommon. Improvement may occur after the lapse of several months, but recurrences especially in the fall and spring are common.

Vegetable Poisoning.—Poisonous symptoms sometimes follow the eating of potatoes that have "sprouted." The symptoms are attributed to solanin, which, under the influence of Bacillus solaniferum, increases from the normal 0.06 per cent. to as much as 0.4 per cent. The symptoms consist of digestive disturbances (pain, nausea, vomiting, diarrhœa), jaundice, headache, chills, fever, and prostration.

Treatment.—The treatment of food poisonings consists primarily in avoiding the source of the intoxication, the proper preservation of food, careful agricultural supervision to avoid contamination of grain, etc., and in the cases of pellagra, etc., removal from the region of endemic intoxication. In the acute ptomaine poisonings the stomach should be washed out, and the bowels thoroughly evacuated by a brisk cathartic; following this the diarrhœa should be controlled by bismuth, lead, opium, etc. Stimulants, such as alcohol and strychnine, are often required. Hypodermoclysis and enteroclysis also may prove useful in reviving a prostrated patient and favoring elimination of the ptomaines. Arsenic and quinine have proved of value in pellagra; tonics also are indicated. Recently, Cole and Winthrop have recommended the transfusion of pellagrinous blood (believed to contain antibodies), or, if this be not obtainable, of normal blood.

SECTION III.

DISORDERS OF METABOLISM.

Pathological Physiology.—The disturbances of metabolism to which the body is subject are many and various. Some of these constitute clinical entities and are discussed under appropriate headings, such as diabetes, gout, obesity, etc.; others in a broad sense include many of the disorders of the ductless glands and of internal secretion, which also are discussed under appropriate headings; others are not so well defined nor so well understood. These have not attained the dignity of clinical entities, being, as a rule, merely more or less obtrusive manifestations of a wide variety of diseased conditions; they are alike, however, in that they lead ultimately to more or less pronounced poisoning of the body, and since the toxic agent is elaborated within the body, they are distinguished as endogenous poisonings or auto-intoxications. In the majority of cases the poisonous agent has not been identified with certainty, so that these auto-intoxications are still the subject of more speculation than of demonstrated fact. Nevertheless, three types of auto-intoxications may be distinguished, although they are not sharply delimitable the one from the other nor from other diseased states, of which often they are merely a manifestation: (1) Gastro-intestinal auto-intoxications; (2) acidosis and acid intoxications; and (3) retention auto-intoxications.

Gastro-intestinal Auto-intoxications.—Gastro-intestinal auto-intoxications conceivably may result from the absorption of: (1) Poisonous intermediate products of digestion; (2) poisonous end products of metabolism that normally are detoxified in the body or excreted therefrom; and (3) certain products of putrefaction. It is difficult, if not impossible to separate these from the results of the activity of bacteria—those normal in the intestine as well as those foreign thereto, since it is not improbable that bacterial activity, of much importance in the orderly performance of some of the intestinal functions, if perverted, may injuriously affect the intestinal enzymes. Abeyance of the detoxifying function of the liver is often, at least in part, answerable for the gastro-intestinal auto-intoxications. The evidence that intermediate and end products of digestion, such as peptones, purins, nucleo-proteins, etc., may be absorbed and give rise to toxic symptoms is very unsatisfactory and inconclusive and for the present may be disregarded. Putrefactive changes in the intestine involve almost solely the aromatic and sulphur

groups of the protein molecule. The aromatic bodies yield indol, phenol, and skatol, which uniting with sulphuric acid, may appear in the urine (indoxyl-sulphate, so-called indicanuria, etc.). This occurs in the event of protein decomposition elsewhere than in the intestine, and it is doubtful whether of itself it gives rise to symptoms; but with reserve it may be interpreted as a measure of intestinal putrefaction: it occurs most rapidly and in greatest amount in obstruction or paralysis of the small intestine. Various other disorders are attributed, with more or less reason, to gastro-intestinal intoxication: senescence and ultimately death (Metchnikoff); the headache and general malaise of chronic constipation; chlorosis (Sir Andrew Clark); pernicious anemia, attributed by William Hunter to oral and gastro-intestinal toxemia; tetany, sometimes associated with gastric dilatation, and due apparently at least indirectly to the absorption of a poison from the stomach; the toxemia and death of high intestinal obstruction (duodenal auto-intoxication, disturbance of the balance of physiological intra-enteric enzymotic balance, Maury); cirrhosis of the liver; chronic disease of the joints; a so-called *intestinal infantilism*, described by Herter and attributed by him to chronic intestinal infection due to the overgrowth and persistence of the bacterial flora of the intestine belonging normally to the nursling period; and a rare form of cyanosis, known as *enterogenous cyanosis*, due in the one series of cases to methemoglobinemia, the result of the absorption of nitrites from the intestine in chronic diarrhœa, and amenable to a milk diet, and due in another series of cases to sulph-hemoglobinemia, the result apparently of putrefaction of the proteins in chronic constipation, and amenable to treatment directed toward the constipation. The *treatment* of these gastro-intestinal auto-intoxications comprises attention to any demonstrable focus of intoxication, such as pyorrhœa alveolaris or other form of oral sepsis, chronic ulceration of the bowel, rectum, etc.; regulation of the diet, especially restricting the amount of meat, and giving in the one case milk, as well as milk soured with the lactic acid bacillus (Bacillus bulgarius); in another case food that leaves sufficient residue to promote free evacuation of the bowels; medicinal and other treatment of constipation, if it be present; the use of so-called intestinal antiseptics, such as salol, β-naphthol, bismuth sulphocarbolate, resorcin, etc.; and high colonic irrigations

Acidosis and Acid Intoxications.—Acidosis is a diseased condition in which organic acids, notably β-oxybutyric acid, diacetic acid, and acetone (the so-called acetone bodies) appear in abnormal amounts in the blood and the urine (ketonuria); acid intoxication is a diseased condition in which in addition toxic symptoms develop. There is still some discussion as to whether these acetone bodies are toxic per se, or whether they exert their deleterious action merely because they are acid, robbing the body of native alkali, diminishing the power of the blood to take up carbon dioxide, and leading to tissue asphyxiation; perhaps both factors are operative in human pathology. These acetone bodies are derived largely, if not exclusively, from the lower fatty acids; perhaps in small

part from the fatty acid moiety in protein, such as leucin. The condition of acid intoxication may be the result of an augmented formation of these acetone bodies, or of a deficiency of bases; and it is found in: (1) Diabetes, of which it forms a conspicuous and serious feature, and under which heading it is discussed; (2) starvation of whatsoever sort: insufficiency of food; inability to take or utilize food, as in stricture or carcinoma of the œsophagus, stomach, or intestine; diseased conditions of the gastro-intestinal tract leading to long-continued rectal feeding, etc.; it is especially likely to supervene if oxidation is deficient and little or no carbohydrate is taken; (3) the febrile state, in which in addition to destruction of the body fat and protein, the supply of food is often insufficient and cannot be properly metabolized; (4) certain ill-understood conditions of the gastro-intestinal tract, usually associated with constipation, in which I have seen it present in high grade; (5) after anesthesia; (6) in young children, and in excessive degree in the recurrent vomiting of unknown cause in children; and (7) in some cases of pernicious vomiting of pregnancy. The *treatment* of the acidosis consists, in brief, in preventing, if possible, the formation of the acetone bodies by a judicious combination of fat, protein, and carbohydrate (discussed under diabetes), by neutralizing the circulating acids (by administering large amounts of sodium bicarbonate or other alkali), and by supplying deficient bases, such as calcium and magnesium.

Retention Auto-intoxications.—To the retention of excrementitious products, auto-intoxications, such as uremia, puerperal eclampsia, and so-called cholemia are often attributed; but the nature of the supposed excrementitious products have not been determined, and there is considerable evidence tending to support the view that in these conditions the detoxifying function of the liver is the major factor at fault: the complete elucidation of the many problems herewith concerned must await future investigations. Asphyxia may be looked upon as a form of auto-intoxication due to the retention of carbon dioxide. The toxemia that follows extensive burns may be due to the retention of toxic substances incapable of elimination by the burned skin, or to the absorption of a toxic substance from the burned areas. Retention of sodium chloride is at least one factor in the production of the œdema of nephritis, and perhaps of other œdemas.

DIABETES.

(*Diabetes Mellitus.*)

Diabetes is a disorder of metabolism, involving chiefly the carbohydrates, and characterized clinically by hyperglycemia, glycosuria, and ultimately ketonuria and acid intoxication.

Etiology.—Diabetes occurs at all ages, but is most common after the fortieth year; almost one-third of the cases develop in the sixth decade, and about one-fifth prior to the thirtieth year; but it may develop in

advanced life as well as in infancy. It is more common in males than in females in the proportion of 3 to 2. In general, it is less common in this country (though increasing) than it is in Europe; it is especially common among Hebrews, and comparatively rare among negroes. It is a disease of the well-to-do, rather than of the poor (in the proportion of 10 to 1, von Noorden). Hereditary influences are active in some cases, diabetes itself, gout, obesity, and neuropathies being common in the antecedents, as well as in the immediate family of diabetic subjects. It is not uncommon in husband and wife (conjugal diabetes), which without much reason has suggested an infectious cause to some observers.

The direct cause of diabetes is not known. An unhygienic mode of life, exposure to wet and cold, overeating, excessive indulgence in carbohydrates and in meat, alcoholism, sedentary habits, prolonged anxiety, mental strain, emotional and other nervous disturbances, etc., have been credited with etiological significance. The combination of sedentary habits, excessive eating (carbohydrates and meats) and drinking (alcoholism), and prolonged mental strain (absorption in business pursuits, large, financial, and other responsibilities) is common in those who develop diabetes. Disorders that are sometimes attended by temporary glycosuria seem sometimes also to lead to true diabetes, such as: pregnancy; infections (diphtheria, scarlatina, malaria, influenza, syphilis, etc.) which perhaps act by setting up pancreatitis; injury and disease of the brain and spinal cord (epilepsy, chorea, organic disease especially, but not alone, of the medulla and the floor of the fourth ventricle), etc.

Pathology.—The important lesions occur in the pancreas—for our knowledge of which fact we are indebted to Minkowski, von Mering, Opie, etc. Cecil recently, studying 90 cases, found lesions of the pancreas in 79 (87.4 per cent.); in these the islands of Langerhans were always, sometimes alone, diseased (sclerosis, hyaline degeneration, leukocytic infiltration, and hypertrophy). 97 per cent. of the cases occurring after the age of thirty years were associated with lesions of the pancreas, and 87 per cent. occurred in association with chronic interacinous pancreatitis accompanying arteriosclerosis. 50 per cent. of the cases occurring before the age of thirty years were associated with lesions of the pancreas; and 75 per cent. of all cases of diabetes in which the pancreas was normal occurred before the age of thirty years. In addition to disease of the pancreas, the liver is usually enlarged; fatty changes are common; there may be cirrhosis, which is usually associated with pigmentation of the liver, pancreas, skin, etc. (hemochromatosis, bronzed diabetes). The nervous system shows divers but inconstant lesions: sclerosis, cysts, or tumors in the medulla and sometimes elsewhere; meningitis and the lesions of different organic diseases; degeneration of the posterior columns of the cord and peripheral neuritis (the cause of so-called diabetic tabes), etc. Usually there is arteriosclerosis and hypertrophy of the heart. Hyperglycemia (as much as 0.5 per cent.

instead of the normal 0.1 to 0.15) is constant, and the opsonic index is reduced. The thyroid, pituitary, and adrenals are sometimes enlarged or otherwise diseased. Tuberculosis, especially of the lungs, bronchopneumonia, croupous pneumonia, chronic nephritis, gangrene, pyococcic infections, such as furuncles, carbuncles, etc., are common complications, and may be the cause of the fatal issue. Chronic nephritis occurs in about one-half of the cases, and is attributable to the polyuria and ketonuria.

The ultimate nature of diabetes is not yet perfectly understood, largely because the fate of carbohydrates in the body in health has not been fully elucidated. It is known that normally the carbohydrates of the food are converted into glycogen and warehoused in the liver and the muscles; and that under some circumstances glycogen may be formed from the non-nitrogenous moiety of the food protein as well as the body protein. As required by the body for heat and energy, the glycogen undergoes destruction, being converted largely into alcohol and carbon dioxide. How this glycolysis is effected is still the subject of study. The tendency of modern studies is to minimize the significance of the liver, which is believed to act chiefly as a storehouse, liberating the stored glycogen as it is required and preventing an excess going over into the circulation at any one time (which would result in hyperglycemia). There is, however, an intimate relationship between the pancreas and the muscles. The islands of Langerhans in the pancreas produce a secretion containing a ferment or zymogen, which, reaching the muscles by way of the blood stream, encounters there another ferment or proferment; an interaction occurring between the two results in the production of another ferment, which has the specific property of destroying dextrose (glucose). In pancreatic diabetes the pancreatic ferment or zymogen is deficient or absent; the muscular ferment or zymogen is not activated, the glycolytic ferment is not formed, and glucose is not combusted, but passes over in the blood in excess (hyperglycemia), and is excreted in the urine (glycosuria). By some investigators it is believed that the pancreas, rather than supplying a substance requisite for the proper metabolism of sugar, has the property of neutralizing a poison which, not detoxified, leads to the accumulation of sugar in the blood. In many cases the ability properly to utilize fats also is more or less impaired, and β-oxybutyric acid and its derivatives (oxidation products), diacetic acid and acetone, intermediary products of fat metabolism (derivatives of the lower fatty acids), are excreted in the urine (ketonuria), and acetone also with the expired air. This occurs especially in conditions of carbohydrate starvation; and, often in the early stages of the disease, at least, can be prevented by feeding carbohydrates, which are concerned in providing the oxygen necessary for the ultimate oxidation of the fatty acids to carbon dioxide and water; later, however, this becomes impossible, and a dangerous acid intoxication ensues and may lead to a fatal termination.

Associated disturbance in the carbohydrate and fat metabolism is

found in some cases of diabetes in the obese, in some of which the conversion of carbohydrates into fat is not seriously disturbed, although the carbohydrate tolerance may be low (alimentary glycosuria, latent diabetes); in some of these true diabetes may subsequently develop. Disorder of the protein metabolism is apparent in the fact that an excess of protein increases the glycosuria in some cases; some observers believe that an excess of protein is at least partly responsible for the disease (said to stimulate the thyroid).

There is in diabetes some relationship between the pancreas and the ductless glands—the thyroid, the adrenals, and the pituitary body. These, particularly the thyroid and the pituitary, may be found enlarged; and diabetes clinically is found associated with more or less well-developed Graves' disease and akromegaly. There is believed to be an antagonism between the pancreas and the thyroid; hyperactivity of the thyroid may produce glycosuria. It has been found also that the injection of adrenalin results in hyperglycemia and glycosuria (adrenal diabetes), and an adrenal influence in human diabetes has been postulated (interference with the oxidizing power of the pancreas, or an action upon the sympathetic nervous system, resulting in the non-warehousing or the release of glycogen, with ensuing hyperglycemia and glycosuria). The administration of phloridzin (a glucoside) also results in glycosuria without hyperglycemia; it is attributed to a toxic influence on the renal epithelium, and is used as a test of the functional capacity of the kidney. The influence exerted by the nervous system in some cases is not well understood. Some observers believe that the disturbance in diabetes is not so much in the pancreas and its internal secretion as in the nerves that pass through the pancreas; others believe that the nervous influence results in the imperfect storage or liberation of glycogen in the body. Hyperglycemia is a constant feature—an increase in the sugar content of the blood from the normal 0.1 to 0.15 per cent. to 0.3 or 0.5 per cent. To it is attributable indirectly the fatigue and emaciation, on account of the calories thus lost. A daily excretion of 100 grams of sugar, representing 400 calories (16 per cent. of the calories required for a man weighing 70 kilograms), must be met by increased protein and fats, whence the hyperglycemia indirectly results in increased appetite.

Symptoms.—In the great majority of cases the onset is insidious and the disease often has made considerable progress by the time obtrusive symptoms become manifest—chronic cases, which may last for years and are especially common in subjects past middle life; rarely the onset is abrupt, or the disorder, having been existent though ill-declared for some time, suddenly (perhaps following an injury, shock, or some emotional disturbance) becomes very acute and rapidly progressive—acute cases, which are especially common in young subjects and may soon lead to death. There is, however, no essential difference between the two classes of cases. As a rule, the early symptoms consist of malaise, readily induced languor, or loss of flesh and strength; or frequent micturition, polyuria, or excessive thirst attracts attention. The fully developed

disease is characterized by hyperglycemia, polyuria, glycosuria, and ultimately ketonuria, acid intoxication, and fatal coma; polydypsia and polyphagia; often but not always marked emaciation; and a number of variable and inconstant symptoms referable to the different systems of organs (complications).

Major interest and significance attaches to the urine—which is increased in amount, pale in color, acid in reaction, of increased specific gravity, and contains sugar, and often one or more of the acetone bodies. The total twenty-four-hour amount of urine averages between three and six liters; but enormous amounts (twenty to thirty or more liters) may be excreted, and in other cases the amount may be scarcely, if at all, above the normal (mild cases; cases markedly improved; under the influence of infectious processes; prior to the fatal issue). The specific gravity varies, as a rule, between 1025 and 1040, but it is sometimes quite low (1015 or less), and rarely it may be very high (1050 to 1070). The sugar content varies—from 0.5 to 2 or 3 per cent. in mild cases, to 5 to 10 per cent. in severe cases. The total amount of sugar excreted daily varies from 50 to 200 grams or thereabout in mild cases to 500 to 1000 grams or more in severe cases; but it is markedly influenced by the diet, the mode of life, the form of treatment, and approaching fatality and coma.

The total nitrogen of the urine (normally 10 to 15 grams daily) is usually increased (15 to 20 grams or more); this is due, as a rule, to the increased protein ingestion, but in severe cases, especially with high glycosuria and during coma, it is due also to destruction of the body protein. The total urea is increased (for the reasons just mentioned); the percentage of urea is usually unchanged in the absence of acidosis; it is diminished when acidosis is present (on account of the increased ammonia). The creatinin may be increased; the uric acid may be normal or increased. Of special importance and significance is the increased output of ammonia. Normally this varies from 0.5 to 1 gram daily, up to 1.5 grams on a meat diet; in diabetes it is often increased to 3 to 6 grams, and may reach 8 to 10 grams or more; and instead of forming 2 to 5 per cent. of the total nitrogen, it may form 10 to 20 per cent. This increase is to be interpreted as an effort on the part of nature to neutralize the existing acidosis—the result of the acid derivatives of the proteins and the fats. The sulphur and phosphorus of the proteins normally are oxidized to H_2SO_4 and P_2O_5, but encountering sufficient fixed bases they are neutralized; when in excess, however, they unite with some of the ammonia (a product also of protein katabolism) and are carried off in the urine. In addition, the acetone bodies, when present, are also, at least in part, neutralized by ammonia. The ammonia excretion thus is a trustworthy indication of the degree of existing acidosis, and is more readily determined. The acetone bodies, β-oxybutyric acid, diacetic acid, and acetone (of which the succeeding is the oxidation product of the preceding) are often present in the urine (ketonuria) in variable amounts. Normally acetone is present in the urine (0.01 to 0.03 gram

in the twenty-four hours); in diabetes this may be increased to 2 or more grams, and the total excretion of acetone and diacetic acid may reach 5 to 8 grams, while that of β-oxybutyric acid may reach 50 to 70 grams. The acetone bodies are derived from the lower fatty acids of the organism and the food, but they occur in the urine only in the event of disordered carbohydrate metabolism, the katabolism of 80 to 100 grams of carbohydrates being ordinarily sufficient to prevent the ketonuria. This rather than the glycosuria is the important index of the severity of the diabetes: the acetone bodies are absent in mild cases for a long time, and acetone is the first to appear; later, diacetic acid is added, and in advanced and severe cases (absolute loss of carbohydrate tolerance, with the excretion in the urine not only of all the ingested carbohydrates, but also of sugar derived from the non-nitrogenous portion of the proteins), all three bodies may be encountered.

Albuminuria is encountered sometime in at least one-half of the cases. It is usually slight in grade, and may be due to local catarrhal disorders of the urinary tract, general infections, arteriosclerosis, degeneration of the renal epithelium (overwork, from long-continued polyuria, glycosuria, and ketonuria); in some cases there may be also casts and other evidences of true nephritis. Albuminuria, previously absent, is likely to appear as a more or less immediate forerunner of coma; indeed, it is not unlikely that some of the cases of coma are uremic rather than diabetic in nature.

Most of the other important symptoms are referable to the digestive tract and the nervous system. Thirst may be the first obtrusive symptom, and is often exceedingly distressing; it is probably due to the systemic demands for fluids; the intake is usually in direct ratio to the output. The appetite may or may not be inordinate. Despite the large amount of food taken, digestion is often good, although indigestion (sometimes associated with gastrectasia) and constipation (sometimes alternating with attacks of diarrhœa) are not uncommon. Despite the large amount of food taken the patient may emaciate rapidly. The tongue is usually dry, red, and glazed, perhaps fissured; the saliva is scanty; there may be aphthous and other forms of stomatitis; and the teeth readily decay. The liver may be enlarged—in ordinary diabetes, as well as in bronzed diabetes (hemochromatosis).

Of the nervous symptoms, pruritus, especially of the genitalia, is often exceedingly distressing. In addition there may be paresthesias, neuralgias, or peripheral neuritis (single or multiple). Bilateral sciatica is common. In almost one-half the cases the knee-jerks are diminished or lost; this may be associated with paresthesias, irregular pains, and neuromuscular cramps in the legs. Some of these cases present a characteristic steppage gait, degeneration of the posterior columns of the spinal cord, perhaps optic atrophy, and other evidence of tabes (so-called diabetic tabes). The cranial nerves (especially the abducens, the oculomotor, the facial, etc.) and the spinal nerves also may be affected (paresthesia, paresis, trophic disturbances, herpes zoster, perforating

ulcer of the foot, etc.). Epileptiform attacks occasionally occur. Impotence (probably due to spinal degeneration) is common, and may be an early symptom. The patient is likely to become nervous, restless, irritable, hypochondriacal, or melancholic.

The most important of the nervous phenomena is coma—which is the direct cause of the fatal issue in almost all of the young subjects and in from one-half to three-fourths of the older subjects (in unusual cases the coma may be not diabetic, but uremic, or due to meningitis, cerebral hemorrhage or softening, etc.). Diabetic coma is attributed to acid intoxication (β-oxybutyric acid), of which it is the final stage, and it is to be feared in all cases in which ketonuria occurs and when the daily output of ammonia (the index of the acidosis) exceeds 3 grams. The coma may develop suddenly in the midst of comparatively good health; or it may be precipitated by severe exertion, excitement, local infection, etc.; or without exciting cause it may come on gradually with weakness, headache, restlessness, anxiety, somnolence, nausea, vomiting, syncope, Kussmaul's dyspnœa, or air-hunger (deep, energetic, long-drawn inspiration, contrasting strongly with the general asthenia, and short, sighing expiration, unassociated with cyanosis, at the beginning at least), a sweetish, fruity odor to the breath (excretion of acetone and diacetic acid with the breath), and gradually deepening coma. The pulse is usually small and rapid, the pupils dilated, the reflexes abolished, and the temperature subnormal; convulsions are rare. Death usually ensues in from a few hours to a few days; rarely the patient may recover, and perhaps succumb to a subsequent attack.

Complications.—Complications are quite common, being due to the hyperglycemia and its attendant impaired nutrition and lessened resistance to bacterial infection, acidosis, etc. Diminished resistance to bacterial infection (lessened opsonic index) is answerable for the common furunculosis, boils, and carbuncles (usually staphylococcic infection), tuberculosis, pneumonia, and bronchopneumonia (perhaps followed by abscess or gangrene), which are responsible for most of the fatal issues not due to coma, but which also may induce the fatal coma. There are many sorts of skin eruption, eczema (set up sometimes by intolerable itching), urticaria, xanthoma, purpura, etc. Gangrene, especially of the leg, is not uncommon in middle-aged or elderly subjects, even in mild cases; it is due to atheroma or obliterating endarteritis.

Generalized arteriosclerosis is common and may be associated with hypertrophy of the heart; rarely, toward the end, in cases of failing heart or advanced nephritis, œdema may supervene, but it is uncommon—on account of the marked polyuria and consequent "dryness" of the tissues. Amenorrhœa is common. Conception may occur, but is likely to be followed by abortion or aggravation of the disease. Vulvitis, vaginitis, urethritis, etc., are common in the female, and balanitis, phimosis, etc., in the male. The special senses may become involved—amblyopia, amaurosis, retinitis, cataract (especially in young subjects), chronic catarrhal deafness (otitis media), etc.

Diagnosis.—The diagnosis comprises not only the mere recognition of the disease and its differentiation from other disorders that resemble it, but also the determination of the type and severity of the disorder, of the degree of carbohydrate tolerance, of the presence and the degree of acidosis, and of the general nutrition of the patient. Polyuria, polydypsia, polyplagia, emaciation, and such suggestive phenomena as genital pruritus, impotence, bilateral sciatica, recurring boils, etc., may suggest the disease; but the diagnosis usually, and in the early stages always, depends upon an examination of the urine. Too much importance cannot be attached to the routine examination of the urine, in all cases of diseases, if the early stages of diabetes are to be diagnosticated.

The carbohydrates include: (1) The monosaccharides or glucoses—glucose (dextrose, grape sugar), levulose (fruit sugar), and galactose (invert milk sugar); (2) the disaccharides or saccharoses—saccharose (cane sugar), maltose (malt sugar), and lactose (milk sugar); (3) the polysaccharides or amyloses—starch, glycogen, dextrin, cellulose, and certain gums; and (4) the pentoses or five-carbon sugars—arabinose and xylose; and glycuronic acid. Of these, only glucose, lactose, maltose, levulose, pentose, and glycuronic acid possess clinical importance in the present connection. Glucose (dextrose) may be determined by Fehling's test, the fermentation test, the phenylhydrazin test, and dextrorotation of polarized light (in the polariscope); but one must also exclude certain non-carbohydrate reducing substances, such as uric acid, creatinin, alkapton, and certain drugs (for details one should refer to books on clinical laboratory diagnosis).

Having determined the presence of glucose in the urine, it is advisable to ascertain whether the condition is: (1) Toxic glycosuria, such as may follow the administration of anesthetics, or certain drugs and poisons, such as phloridzin, chloral, morphine, coal-tar products, etc., or develop in the course of or following pregnancy, some infection, etc.; (2) neurotic glycosuria, such as may be due to divers emotional or other disturbances, shocks, etc.; or occur in association with different nervous diseases; (3) alimentary glycosuria (the inability to metabolize 100 grams of glucose taken at one time without the appearance of glucose in the urine); or (4) true diabetic glycosuria. The toxic, neurotic, and alimentary cases are transitory and intermittent; as a rule, the amount of sugar excreted is small, and the condition may entirely disappear: but many of the cases bear a more or less intimate relationship to true diabetes, of which they may be an early stage or ill-developed form, and to which they may progress.

From a diagnostic, prognostic, and therapeutic standpoint it is important in the case of true diabetes (continuous glycosuria associated with the other mentioned symptoms) to determine the type, whether mild, intermediate, or severe; that is, to determine the carbohydrate tolerance. Mild cases are those in which some degree of carbohydrate tolerance is maintained; that is, 100 grams of glucose can be metabolized without the development of glycosuria, and no disturbance

in fat metabolism is present; that is, there is no ketonuria. Intermediate cases are those in which the carbohydrate tolerance is almost if not quite lost (less than 30 to 50 grams); that is, the urine becomes aglycosuric on a carbohydrate-free diet continued for a week or more; and there is also some disturbance of fat metabolism, shown in acetonuria and diaceturia from time to time, but especially when the patient is on a carbohydrate-free diet. Severe cases are those in which the carbohydrate tolerance is entirely lost; that is, the urine does not become aglycosuric, even when a carbohydrate-free diet has been continued for several weeks, although aglycosuria may be produced by restriction of the protein intake; and ketonuria (including often β-oxybutyric acid) is a constant feature. The severity of the case is to be determined not so much by the output of glucose, assuredly not by the percentage output, as by the output compared with the carbohydrate intake. In some cases of total carbohydrate intolerance sugar may be found in the urine on a fat-protein diet in the proportion of 3.65 to 1 of nitrogen (the fatal ratio, Mandel and Lusk). The majority of cases of diabetes are mild, but they are most common in elderly and obese subjects; intermediate cases are common in middle life; severe cases are most common in youthful and in thin subjects.

The acidosis may be determined by Gerhardt's, Arnold's, or Hart's tests (for diacetic acid and acetone), by the Huppert-Messinger quantitative test, by levorotation of polarized light after complete fermentation of the urine, by determining the ammonia output, and roughly by the amount of sodium bicarbonate required to render the urine alkaline (normally 5 to 10 grams, Blum). The general condition of the patient is to be determined by repeated physical examination and determination of the body weight. Diabetic coma may be distinguished from uremic coma by ketonuria, perhaps glycosuria, fruity odor to the breath, and the characteristic Kussmaul dyspnœa; albuminuria and the cardiovascular and other signs of chronic nephritis are often present, and may at first be confusing.

Prognosis.—The course of the disease is rarely acute (several weeks); usually it is chronic—several years to many years (twenty or more). The prognosis varies with the severity of the case: it is bad in youthful subjects (in infancy, childhood, and adolescence); in thin rather than obese subjects; in the event of marked and rapid loss of flesh (despite the ingestion of a large amount of food); in the event of carbohydrate intolerance, marked glycosuria, ketonuria, non-response to treatment, and infectious complications. Coma is almost invariably fatal. The prognosis is favorable in elderly obese subjects; in cases with fair or moderate carbohydrate tolerance, slight glycosuria, and no ketonuria; in the absence of complications; and in the event of response to treatment (lessening of glycosuria and increase in weight).

Treatment.—The treatment is almost, if not quite, wholly dietetic. General hygienic measures; a life of comparative ease and quiet, free from undue professional or business exactions, worry, and emotional and other disturbances; a moderate amount of regulated exercise (which in some,

especially mild, cases appears to increase sugar metabolism, but which may have a contrary effect in severe cases); now and then a course of treatment at some one of the well-known spas; and drugs—each has its place in treatment, but the physician must individualize; he must treat the patient, not the disease.

DIET.—Since the patient has more or less lost the ability to metabolize carbohydrates, and we are unable directly to influence the carbohydrate metabolism, it becomes necessary to nourish him largely on a fat-protein diet. The difficulty in doing this is apparent from the fact that a healthy subject ordinarily derives almost one-half of his heat and energy from carbohydrates; that this is largely or wholly lost to the diabetic through the glycosuria; that it is difficult to supply this deficiency with a fat-protein diet (which is not the most palatable); and that continuation of the hyperglycemia results in a decreased tolerance of the tissues for carbohydrates—as, on the contrary, lessening of the hyperglycemia results in an increased tolerance; and that a carbohydrate-free diet is likely to lead to a dangerous acid intoxication. The permissible and the prohibited foods are mentioned in Table I, which as well as the other tables appended have been arranged by T. C. Janeway.

In the treatment of every diabetic subject the first essential is to deter-

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TABLE I.—General Diabetic Diet List.

May take freely: Soups—all meat soups and broths; may add vegetables allowed, egg, or cheese. Meats—all fresh, smoked, or cured meats (except liver), poultry, and game, without sauces or gravies containing flour; pate de foie gras. Fish—all kinds of fish, except oysters, clams, and scallops, cooked without bread crumbs or meal; dried, salted, smoked, or pickled fish. Eggs—prepared in any way without flour. Fats—butter, lard, suet, olive oil, or other fats. Cheeses—all kinds, especially cream, Swiss, English, and pineapple cheese. Vegetables and salads—asparagus, beet greens, Brussels sprouts, cabbage, cauliflower, celery, chicory, cresses, cucumbers, egg-plant, endive, kohl-rabi, leeks, lettuce, okra, pumpkin, radishes, rhubarb, salsify, sauerkrout, spinach, string beans, tomatoes, vegetable marrow. Pickles made from the above-mentioned vegetables, unsweetened; ripe olives. Fungi—mushrooms and truffles. Cream—not over three ounces a day. Condiments—salt, pepper, cayenne, paprika, curry, cinnamon, cloves, English mustard, nutmeg, caraway, capers, vinegar, and the piquant sauces in small quantities, unless specially forbidden. Desserts—jellies made from gelatine; custards and ice cream made with eggs and cream; all sweetened with saccharin and flavored with vanilla, coffee, or brandy. The following recipes for desserts are suggested: Baked custard—1 egg, 1½ ounces of cream, 2½ ounces of water; two or three ½-grain saccharine tablets, 8 drops of vanilla essence; beat up well, pour into a buttered dish, grate a little nutmeg on top, and bake twenty minutes. Coffee ice cream—1½ ounces of cream, 1½ ounces of water, 1 ounce of strong coffee, two or three ½-grain saccharine tablets; dissolve; add 1 egg, well beaten; mix in a saucepan and beat slowly with stirring until it thickens; set aside until cool; then freeze. Nuts—butternuts. Beverages—tea or coffee, sweetened with saccharin, and with the portion of cream allowed; whiskey, brandy, rum, and other distilled liquors up to 3 ounces a day; light Rhine or Moselle wine, claret, or Burgundy up to 16 ounces a day; mineral waters of all kinds; lemonade in small quantity, sweetened with saccharin.

Articles prohibited, except as prescribed in the accessory diet (carbohydrate equivalents, Table IV): Sugars and sweets of every kind; pastry, puddings, preserves, cake, and ice cream; bread and biscuit of all kinds, toast, crackers, and griddle cakes; cereals, such as rice, oatmeal, sago, hominy, tapioca, and barley; macaroni, potatoes, carrots, parsnips, beans, peas, beets, green corn, and turnips; fruit of all kinds, fresh and dried; soups, sauces, or gravies thickened with flour or meal, or made with milk; beer, ale, porter, all sweet wines, sherry, or port wine, sparkling wines, cider, and liqueurs; milk, chocolate, or cocoa; all sweet drinks and soda water,

mine the carbohydrate tolerance and the degree of acidosis (if there be any); the carbohydrate tolerance should be redetermined at least every two months in mild and intermediate cases, and perhaps more frequently in the severe cases. To determine the carbohydrate tolerance, the patient should be put upon a fixed fat-protein diet of sufficient caloric value to maintain strength and body weight; that is, 35 large calories per kilogram of body weight (a man weighing 70 kilograms—150 pounds—requires about 2500 calories per day). The carbohydrates should be withdrawn gradually (three to seven days, depending upon the severity of the case), since sudden withdrawal may result in a severe, perhaps fatal, acidosis. While determining the carbohydrate tolerance, the presence and the degree of acidosis should be inquired into, and if present the patient should be given one-half to one ounce of sodium bicarbonate daily. A suitable fat-protein diet is indicated in Table II. To this fat-protein diet 100 grams (3 ounces) of white bread (55 per cent. of carbohydrate) in three equal portions should be added. The bread should be gradually withdrawn until the urine has become sugar-free; if the patient is not aglycosuric when the bread has been entirely withdrawn, the diet without bread should be continued for one week or more, in the hope that the urine will become sugar-free. If the

TABLE II.—Standard Strict Diet.

Breakfast: Coffee with 1½ ounces cream; 2 eggs cooked with ½ ounce butter; 3 ounces ham.

Luncheon: Bouillon with 1 raw egg; 3 ounces sirloin steak, chicken, or leg of lamb; 1 ounce bacon; vegetable from list, 2 tablespoonfuls, with ½ ounce butter; dessert made with 1 egg and 1½ ounces cream; 6 ounces wine, or 1 ounce whiskey or brandy.

Afternoon tea with ½ ounce cream.

Dinner: Any clear soup; 3 ounces fish (salmon, shad, or mackerel), with ½ ounce butter; ¼ pound roast pork, beef, mutton, turkey, or lamb chops; vegetables from list, 2 tablespoonfuls, with ½ ounce butter; salad with ½ ounce oil in dressing; 1 ounce cheese, English, pineapple, Swiss, or full cream; 6 ounces wine, or 1 ounce whiskey or brandy; demitasse of coffee.

Protein	= 126 grams;	515 calories
Fat	= 222 grams;	2065 aalories
Carbohydrate	= 15 grams;	60 calories
Alcohol	= 30 grams;	210 calories
		2850

TABLE III.—Standard Diet with Restricted Protein.

Breakfast: Coffee with 1½ ounces cream; 2 eggs with ½ ounce butter; 1 ounce bacon.

Luncheon: 2 eggs; 1 ounce bacon; 2 ounces lamb chops (1), ham (2), beefsteak (3), chicken (4), or fish (5) broiled with ½ ounce butter (each day select meat with same number for luncheon and dinner); vegetable from list, 2 tablespoonfuls, with ½ ounce butter; dessert made with 1 egg and 1½ ounces cream; 6 ounces wine or 1 ounce whiskey or brandy.

Afternoon tea with ½ ounce cream.

Dinner: Any clear soup; ¼ pound roast pork (5), beef (4), mutton (3), turkey (2), chicken (1), or lamb (1); (each day select meat with same number for luncheon and dinner); vegetables from list, 2 tablespoonfuls, with ½ ounce butter; salad with ½ ounce oil in dressing; 1 ounce cream cheese; 6 ounces wine or 1 ounce whiskey or brandy; demitasse of coffee.

Protein	= 82 grams;	334 calories
Fat	= 215 grams;	2008 calories
Carbohydrate	= 15 grams;	60 calories
Alcohol	= 30 grams;	210 calories
		2612

urine becomes sugar-free, gradually increasing, weighed amounts of white bread may be added to the diet until sugar is again excreted. Should the patient continue to excrete sugar on the fat-protein diet (without bread), the protein intake must be reduced, since considerable protein intake increases the glycosuria. A suitable diet with restricted protein is that of Table III.

The foregoing procedures will determine the carbohydrate tolerance (expressed in weighed amounts of white bread), as well as the presence or absence of acidosis, and permit of a classification of the case—mild, intermediate, or severe. As regards the acidosis, subjects who tolerate 100 grams or more of bread rarely have acidosis; those who tolerate less carbohydrate may or may not exhibit ketonuria, but the ketonuria that develops during this experimental withdrawal of carbohydrates lasts only a few days, as a rule; the diacetic acid disappears, and the amount of acetone is not in excess of the normal.

The subsequent treatment of the patient varies with the type of the disease, but the main indications are to maintain nutrition and body

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TABLE IV.—Table of Carbohydrate Equivalents.

Article.	Per cent. carbohydrates.	Amount in ounces equal to 1 ounce white bread.
Breads: White	51–55	1
All other	47–53	1
Rolls and biscuit	52–60	1
Cornbread	46	1⅛
Crackers, average	69–72	¾
Cereals: Oatmeal, boiled	11.3	5
Hominy, boiled	17.8	3
Macaroni, boiled	15.8	3¼
Rice, boiled	24.4	2⅛
Tubers and Legumes: Potatoes, cooked	18–20	3
Parsnips	13	4
Beans, baked	20	2¾
Beans, lima, cooked	20	2¾
Peas, green, cooked	15	3½
Milk	4–5	10
Fruits: Apples, apricots, and pears	12–14	4
Cherries	15	3½
Huckleberries	16	3¼
Plums	20	2½
Bananas	22	2½
Nuts: Filberts	12	4½
Almonds	15	3½
Peanuts	22	2½

TABLE V.—Green Days.

Breakfast: 1 egg, boiled or poached; cup of black coffee.

Dinner: Spinach with a hard-boiled egg; ½ ounce bacon; salad, with ½ ounce oil; 6 ounces of wine or 1 ounce of whiskey or brandy.

4.30 P.M.: Cup of beef tea or chicken broth.

Supper: 1 egg, scrambled, with tomato and a little butter; ½ ounce bacon; cabbage, sauerkraut, string-beans, or asparagus; cup of tea; or white wine, 4 ounces; or whiskey or brandy, 1 ounce.

½ ounce of sodium bicarbonate in the twenty-four hours.

Carbohydrates = about 5 grams. Calories = 575.

weight, to lessen the hyperglycemia (of which the glycosuria affords a clinical measure), and to prevent or control the acidosis. Mild cases, in which the carbohydrate tolerance is 100 grams or more of white bread, should be kept within their limit. Physicians who prefer gluten bread should not prescribe it under the misapprehension that it is harmless; most of those on the market contain from 30 to 70 per cent. of carbohydrate; ordinarily it is better to give a weighed amount of white bread of known carbohydrate content. The patient may be permitted to vary the carbohydrates of his diet in accordance with his desires and his metabolic activities—which in individual cases is not alike for all carbohydrates. Table IV shows some permissible carbohydrates and their equivalents in terms of white bread. In the intermediate cases, the diet in general should conform to that of Table II, with the addition of such small amount of carbohydrate as may be found to be within the patient's capabilities to metabolize. Considerable bacon, fat meats (fat beef, pork, mutton, etc.), and fish (salmon, etc.), up to a total of not more than 150 grams (5 ounces) daily, butter, thick cream, cheese, oil, etc., are especially to be used. The digestion of the considerable amount of fat and the prevention of diarrhœa from imperfect digestion of fat may be facilitated by a moderate amount of alcohol, as suggested in Tables I, II, and III. In severer cases the difficulties are increased; the protein must be reduced, and one may not add carbohydrates. Study and rearrangement of the diet, however, will permit one to nourish the patient sufficiently on a very low protein diet (100 grams or less). Idiosyncrasies, however, must be met—some patients cannot, others will not, adhere to the prescribed diet, others fail in nutrition, or acidosis develops, in which cases, heterodoxical as it may seem, it is better that the patient have a little sugar in his urine than that he fail in flesh and strength or run the risks of acid intoxication. Now and then a diet containing very little protein is serviceable: largely broths (the hunger days of Naunyn), or green vegetables, eggs, bacon, butter, black coffee, and alcohol (the green days of von Noorden, Table V).

Good results have been reported from time to time by a milk cure, Mosse's potato cure, and von Noorden's oatmeal cure. The last-mentioned consists of 250 grams of oatmeal, salted to suit the taste, and three to four quarts of water, cooked for two hours on a moderate fire; thereupon 250 to 300 grams of butter and 100 grams of a vegetable albumin, such as roborat, gliden, rice albumin, etc., is added (six to eight eggs may be substituted for the albumin in the country). This is to be taken in divided portions during the twenty-four hours, with a little black coffee or alcohol. Von Noorden recommends the treatment in cases of moderate severity, four or five times a year, for a week—thus: one vegetable day, three oatmeal days, two vegetable days. The return to the usual diabetic diet must be gradual, since otherwise increased glycosuria and coma may ensue.

Drugs.—The drug treatment of diabetes is notoriously inefficient, although drugs have their place in the treatment; but without proper

dietetic regulation drugs are worse than useless. Opium, codeine, and arsenic often seem to be of much service; they appear to favor oxidation, lessen glycosuria (perhaps by preventing protein destruction), and increase the general well-being of the patient. The bromides are of service in many of the nervous manifestations. Tonics, such as iron, quinine, strychnine, etc., are often useful. Jambul, chimophila, the salicylates, etc., have been extolled from time to time. The alkalies are of distinct service—the alkaline mineral waters and the carbonate, bicarbonate, citrate, tartrate of sodium. In the event of acetonuria one-half to one ounce of sodium bicarbonate should be given daily, and twice or thrice as much in impending coma. Developed coma is usually fatal, but it is sometimes relieved by intravenous injections of 2 to 4 per cent. of sodium carbonate in physiological saline solution. The complications are to be treated on general principles.

DIABETES INSIPIDUS.

(*Polyuria.*)

Diabetes insipidus is a chronic disorder characterized by the passage of large amounts of non-saccharine and non-albuminous urine of low specific gravity.

Etiology.—The disorder is most common in males between the twentieth and the thirtieth year; more than half of the cases occur between the tenth and the fortieth year. Some cases seem to be hereditary, or several cases occur in the same family. Two types of polyuria may be differentiated—the secondary or symptomatic and the primary or idiopathic. Symptomatic polyuria is especially common: (1) In diabetes, chronic interstitial nephritis, after drinking large amounts of water, etc., in organic and functional disorders of the cerebrospinal and sympathetic nervous system, such as trauma, syphilis (gumma, basilar meningitis), tumors (especially of the floor of the fourth ventricle), hemorrhage, hydrocephalus, lesions of the medulla, of the trapezoid body of the pons, or of the middle cerebellar lobe, etc., fright, emotional disturbances, hysteria, neurasthenia, psychoses, epilepsy, alcoholism, etc.; (2) during or after infections (typhoid, typhus, malarial or scarlet fever, tuberculosis, diphtheria, measles, etc.); and (3) in conditions of general ill health from overwork, mental anxiety, poor nutrition, etc. The idiopathic cases are those that develop without obvious or discoverable cause.

The nature of the disease is not well understood. The basis of it is believed to be a functional incapacity of the kidneys (unassociated with noteworthy structural alterations), due to disturbance of the nervous or vascular mechanism (vasodilatation unaccompanied by rise of the general arterial pressure), in consequence of which the kidneys are unable to excrete urine containing a normal percentage of solids; the normal amount of solids can be excreted only in association with the passage of a large amount of urine, in the absence of which concentration of the blood occurs. Increase in the protein intake does not cause, as it

does in health, an increase in the specific gravity of the urine, but an increase in the amount of urine, the proportion of urea remaining constant. Similar results follow variations in the sodium chloride intake. Occasionally diabetes insipidus is said to develop into diabetes mellitus, and vice versa, but usually this is a misinterpretation of surface phenomena.

Pathology.—There are no constant or characteristic lesions. Divers lesions of the brain are found in about one-third of the cases. The kidneys may be enlarged and congested, and the renal pelves, ureters, and bloodvessels dilated and hypertrophied.

Symptoms.—As a rule, the onset of the disease is insidious; in some cases, as after a fright, etc., there may be a sudden, usually transitory polyuria; in other cases, as after trauma, the onset may be abrupt, but the course protracted. The characteristic phenomenon is the passage of a large amount (5, 10, 20, or more liters daily) of urine, which is pale in color and of low specific gravity (1005 to 1001 or less); the total solids are about normal; albumin and sugar are absent, except in rare circumstances, and then are present only in traces and temporarily. Thirst is an almost constant symptom and may be the first noteworthy manifestation; it is the result and not the cause of the polyuria. The appetite may be poor, normal, or excessive; the stomach may become dilated; the mouth and the skin usually are dry; constipation is common. Headache, nervousness, irritability, loss of sexual power, and other nervous manifestations may occur, the general health may remain for a long time unaffected (depending upon the actuating cause); but emaciation and loss of strength sometimes supervene.

Diagnosis.—The diagnosis is easy. Symptomatic cases should be readily recognized by attention to the causative factors; the frequency of syphilis as an etiological factor should be borne in mind. Diabetes mellitus may be excluded by the absence of hyperglycemia, glycosuria (and high specific gravity), carbohydrate intolerance, ketonuria, and the common complications. Chronic interstitial nephritis may be excluded by the absence of albuminuria, casts, and the cardiovascular, retinal, and uremic manifestations of nephritis; in addition, the specific gravity of the urine is usually higher and the amount less than in diabetes insipidus.

Prognosis.—The duration and prognosis vary with the cause and type of the disease. Recovery, except in the obviously transitory cases, is unlikely, but the patient may live for many years. Death often ensues from complicating infections.

Treatment.—As low a protein and sodium chloride intake as is consistent with the maintenance of strength and weight should be given. It is not necessary or advisable to restrict the fluid intake, since it results in retention of the urinary solids and other harmful consequences. The iodides are often of marked benefit even in apparently non-syphilitic cases. Strychnine and vasoconstrictors, such as ergot, belladonna, etc., are often followed by benefit; adrenalin hypodermicly or intravenously may be tried. Opium and valerian also are of value in some cases.

GOUT.

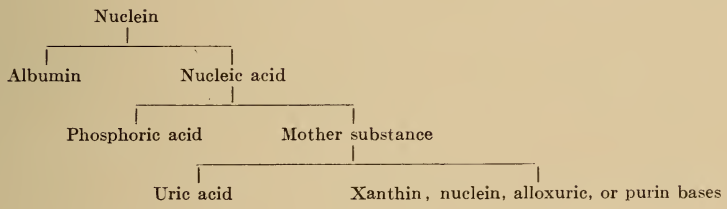
(*Podagra.*)

Gout is a chronic disorder of metabolism attended by marked excess of uric acid in the blood, and the deposition in and about the joints, as well as in other cartilaginous and fibrous tissues of the body, of crystalline sodium biurate.

Etiology.—While the exact disturbances of metabolism occurring in gout are still the subject of much study and discussion, certain predisposing factors are commonly recognized. Of these, heredity is probably the most important. Although it is generally conceded that gout may be acquired, that is, that it occurs in the absence of gouty ancestry, and although not all descendants (children and grandchildren) of gouty subjects become gouty, the common opinion is that at least 50 to 60 per cent. of gouty patients suffer for the sins of their ancestors. Futcher, however, who states that gout is much more common in this country than is generally believed, being often unrecognized, believes that it is much more frequently acquired than inherited, since not more than 31 per cent. of his patients had gouty ancestors. Gout is much more common in males than in females, and transmission is through the male rather than the female members of the family. In the female the disease is almost always inherited. It is more common in the white than in the colored race. It usually makes its appearance during the fourth decade, and then continues more or less through the remainder of life; it may however, develop later, and rarely earlier, sometimes during the second, and even the first decade. Continued overindulgence in alcoholic beverages is an important etiological factor, fermented liquors, such as beer, ale, and porter, and wines, such as sherry, port, and champagne, being much more injurious than distilled liquors, such as whiskey, brandy, gin, rum, etc. Something more than the mere percentage of alcohol seems to be of significance. Overeating combined with insufficient exercise strongly conduce to gout; and it is more the mere overeating as such, rather than the special kind of food (such as red meats, for instance, long but perhaps erroneously too much blamed), that does the harm. Gout is by no means confined to the wealthy and idle class, since "in England the combination of poor food, defective hygiene, and an excessive consumption of malt liquors makes 'the poor man's gout' a common affection" (Osler). Chronic lead poisoning is also an important etiological factor, though less so in this country than in England; how it acts, whether by causing arteriosclerosis, by lessening the alkalinity of the blood (most saturnine patients seem to have an excess of uric acid in their blood), or by provoking trophic changes through the medium of the nervous system, is not definitely known.

Despite considerable patient inquiry and investigation, the ultimate factor in the disorder of metabolism provoking the gouty manifestations has not yet been definitely determined. It is generally conceded that

gout results from faulty metabolism (largely defective oxidation) of the nitrogenous foodstuffs and of the nitrogenous products of tissue waste, and that, in some way or other disturbances in the formation or elimination (or both) of uric acid is, directly or indirectly, in large part at least, the proximal causative factor. Uric acid ($C_5H_4N_4O_3$), as well as other nitrogenous compounds, xanthin, hypoxanthin, adenin, and guanin, are derived from the nuclei of the leukocytes and other cells of the body (endogenous purin bodies) and from the nuclei of ingested food (exogenous purin bodies). Closely related to these are other substances (containing carbon, hydrogen, nitrogen, and oxygen (?)), known as heteroxanthin, paraxanthin, episarkin, carnin, and epiguanin. Collectively, that is, including uric acid, they are known as the xanthin bodies, the nuclein bodies (because of their derivation), the alloxuric bodies (because they contain an alloxan and a urea nucleus), and the purin bodies (because they are derived from or may be prepared synthetically from purin, $C_5H_4N_4$); excluding uric acid, they are known as the xanthin, nuclein, alloxuric, or purin bases. Their derivation may be thus represented graphically:



How uric acid circulates in the blood under normal conditions is not definitely known. It is said to form three groups of salts, which, with sodium as the base, are as follows: (1) Neutral sodium urate, merely a laboratory compound; (2) acid sodium urate, or sodium biurate, which does not occur physiologically, though this is the form in which the uric acid is deposited in the gouty tophi; and (3) sodium quadriurate, which is loosely combined, readily soluble, and the form in which Sir William Roberts and many others believe the uric acid circulates in the blood. Minkowski believes that the uric acid circulates in combination with the purin base, nucleotin-phosphoric acid. In health an adult of average weight on a mixed diet excretes from 0.4 to 1 gram of uric acid, approximating 0.7 gram on an average; and the ratio of uric acid to urea, though it varies much, averages from 1 to 50 to 1 to 70.

In gout, according to Minkowski: (1) The daily excretion of uric acid in the intervals between the acute attacks ranges within the same limits as does the excretion in healthy individuals; (2) in chronic gout, even in those cases in which there is marked deposition of biurates in the tissues, a constant variation from the normal amount of uric acid excretion in any one direction has not been definitely proved; and (3) immediately preceding an acute attack there is regularly a diminution in

the amount of uric acid eliminated in the urine, whereas during and after the attack the uric acid output is increased. Futcher disagrees with these statements in having found almost always a marked diminution in the uric acid excretion in the intervals between acute attacks in chronic tophaceous gout. The blood in gout contains a marked excess of uric acid, and although there has been and still is much discussion as to whether this is due to increased formation, diminished destruction or oxidation, or diminished excretion by the kidneys, the weight of opinion recently tends to support the view that it is due to deficient excretion by the kidneys, and this in turn has been attributed to "functional" disturbances independent of apparent organic disease, to the nephritic lesions so common in gout, and by Minkowski and His to the circumstance (not proved) that the uric acid in gout may circulate in combination different from that of health and more difficult of elimination by the kidneys.

Heretofore too little attention has been paid to the excretion of the purin bases in gout. Since it has been shown that normally about nine-tenths of the purin bodies eliminated is uric acid, and about one-tenth the purin bases, and since there is no evidence that this relationship is disturbed in gout, it is not unlikely, as maintained by Kolish, that at least certain of the gouty phenomena may be due to these purin bases. As regards the phosphoric acid elimination, Futcher has shown that in the intervals between the attacks in chronic gout it falls far below the lower limits for the normal, and that with the onset of the acute attack, concurrently with the increase in the uric acid elimination, there is a parallel rise of the phosphoric acid elimination to the average or even to the upper limits for the normal. Others, however, have obtained somewhat divergent results.

Pathology.—In addition to a marked increase of the uric acid in the blood, a constant feature of the disease first pointed out by Garrod, the joints present the most characteristic lesions of the disease—the deposition of crystalline sodium biurate in the cartilages, synovial membranes, tendons, ligaments, and bursæ. The metatarsal-phalangeal joint of the great toe is most frequently involved, and the lesions may remain confined to this joint for a long time; later, however, the metatarsal, the ankle, the small joints of the hands and fingers, and the knee and the wrist may become involved, but sometimes the lesions affect only the lower extremities. Similar deposits occur also in the helix of the ear, as well as in the cartilages of the nose, eyelids, and larynx; they have been observed in the periosteum (over the sacrum and elsewhere), and in the tendons (the tendons of the hand and forearm, Achilles tendon, plantar surface of the foot, etc.). The deposits of sodium biurate occur in, rather than on, the surface of cartilage, being always covered by a thin layer of cartilage, through which they present as white, "chalky" deposits, or tophi. Whether there is a primary tissue necrosis with a secondary deposition of the sodium biurate, as maintained by Ebstein, or vice versa, as maintained by His and Mordhorst, has not been definitely determined. Ebstein

maintains that the tissue necroses are always primary, that he has found them without any uratic deposits, and that they are acid in reaction—whence the ready deposition of the soluble and loosely combined quadriurate as the insoluble crystalline biurate. Sir William Roberts believes that the deposits occur especially in the cartilages, because in them the nutritional currents are slowest and least abundant, the temperature lowest, and in the articulating surfaces the percentage of sodium chloride highest. As the disease advances the periarticular tissues (ligaments and fibrous tissues) become invaded, and often large tophi or "chalk stones" are formed, which, together with the fibrous thickenings, lead to fixation and much distortion of the joints. Though usually covered by skin, the tophi may cause ulceration of overlying structures, and small, nodular, "chalky" masses may be discharged externally. True bony outgrowths are found about the margins of certain joints in far advanced cases.

The kidneys show the changes of chronic interstitial nephritis (the gouty kidney) or sometimes the type described as the arteriosclerotic kidney. Whitish streaks, evidences of deposits of sodium biurate, may be found in the papillæ, and in and between the uriniferous tubules. General arteriosclerosis is common; indeed, gout is one of the most frequent causes of arteriosclerosis. Hypertrophy of the left ventricle is a usual concomitant. Not infrequently myocardial degeneration leading to dilatation of the heart follows, and is quite often associated with unusual atheroma of the arch of the aorta and the orifices of the coronary arteries. Deposits of sodium biurate have been found in the heart valves. Gouty pericarditis sometimes occurs toward the close of life, and may be the direct cause of death. Emphysema of the lungs is common, and chronic bronchitis and asthma may supervene. Crystals of sodium biurate may be found in the sputum. Neusser's perinuclear basophilic granules are now known to be artefacts. Despite the chronic course of the disease, no noteworthy anemia, as a rule, develops.

Symptoms.—Three varieties of gout are usually distinguished, acute gout, chronic gout, and irregular gout.

Acute Gout.—Acute gout may be abrupt on onset, but, as a rule, it is preceded for several days, or at least a few hours, by vague pains in the joints or muscles, irritability of temper, mental depression, insomnia, dyspepsia, and scantiness of urine (containing a deficiency of uric acid). Occasionally, however, such prodromal manifestations are absent. The attack usually begins abruptly in the early morning hours, the patient being awakened by a sharp excruciating pain in the ball of the great toe. The joint swells rapidly, and becomes red, hot, extremely tender, and the surrounding skin œdematous and shiny. Fever, 102° to 103° F., usually, but not always, accompanies the attack. As the morning wears on the symptoms abate, and the patient may become tolerably comfortable; but the morning of the succeeding day, usually of each succeeding day for a week or thereabouts, witnesses a recurrence of the severe symptoms, which, however, progressively diminish in intensity. Just prior to and

during the early part of the attack the uric acid output is diminished, but later and for a short time after the attack it becomes increased. In addition, the urine is scanty, acid in reaction, of increased specific gravity, and heightened color, and it may contain a trace of albumin or of sugar (transitory). Following the subsidence of the acute manifestations, the patient soon regains his former health—indeed, he may feel much improved. The affected joint soon returns to its normal condition, except in cases of chronic gout, in which deformity sooner or later ensues. The acute attack does not always involve the great toes; occasionally other joints, such as the wrist, knee, or the small joints of the fingers may suffer; and the lesions are not always confined to a single joint, both great toes, for instance, being occasionally affected. The attacks recur at varying intervals—weeks, months, or even years—but with the lapse of time the intervals, as a rule, become progressively less, and the patient gradually develops chronic gout.

Occasionally the local manifestations of gout disappear or improve rapidly, and concurrently herewith serious symptoms referable to the internal organs develop—a condition to which the term *retrocedent* or *suppressed gout* is applied. The symptoms may be gastro-intestinal—abdominal pain, vomiting, diarrhea, with weakness, faintness, and a rapid, feeble pulse, progressing occasionally to death; or they may be cardiac—precordial pain, dyspnea, and palpitation (doubtless in some cases true angina pectoris); or they may be cerebral—mental excitement or depression, apoplexy, delirium, or coma—in many cases probably uremic.

Chronic Gout.—In consequence of repeated attacks of acute gout, commonly with gradually decreasing intervals, the symptoms, though less acute, become more continuous. Urates become deposited in the articular cartilages, later in and about the ligaments and periarticular fibrous tissues; these tophi, which often become extreme, together with fibrous thickenings, occasion fixation and marked swelling and deformity of the joints. Usually the feet are first involved; later the hands, fingers, wrists, knees, etc., may show most extensive changes. As already observed, the tophi may ulcerate through the skin, and chalky deposits may be exposed or even discharged externally. Similar deposits are found in the cartilages of the ear, and elsewhere in the tendinous and fibrous tissues throughout the body. The patient is usually dyspeptic. Gradually arteriosclerosis develops, and the patient presents the common cardiac and vascular manifestations of this condition. Soon also the evidences of chronic interstitial nephritis present themselves: the urine increases in amount, is of low specific gravity, and contains a slight amount of albumin and a few hyaline casts. Aside from the incapacities attendant upon the joint deformities, the patients often exhibit unusual physical and bodily vigor for a long time. The chronic course of the disease is sometimes interrupted by acute exacerbations of unusual severity. The final end is not infrequently determined by inflammations of any of the serous membranes, or by uremia.

Irregular Gout.—Irregular gout is a term applied to an ill-assorted group of symptoms that are found in persons who once have had gout, or who, though they may never have suffered from gout, yet are predisposed by heredity or other factors to its development: a condition to which the term gouty diathesis or lithemia is often applied, but which more than anything else represents a hodge-podge of diagnostic errors. Thus, there may be manifestations of catarrh of the gastro-intestinal tract, and the gouty stomach, the torpid liver, and biliousness are favorite ailments of certain physicians skilled in their treatment. In other cases the urine is lessened in amount, high colored, and uric acid crystallizes out on standing—a circumstance to which undue importance has been attributed. Other disorders ascribed to the same condition comprise eczema, headaches, paresthesias, itching of the palms of the hands and the soles of the feet, cramps in the legs, itching eyeballs, conjunctivitis, iritis, retinitis, tonsillitis, etc.

Diagnosis.—Typical acute gout can scarcely be mistaken for anything else. In atypical cases, as, for instance, when the feet are not involved, or when there is polyarthritis, the differentiation from rheumatic fever may be determined by a tophus in the ear, but the mode of life, the occupation, hereditary factors, the absence of fever, and the condition of the urine may be of contributing diagnostic importance. Systematic search for tophi in all cases would prevent many diagnostic blunders, but small fibroid nodules, Woolner's tip, or small sebaceous cysts should not be mistaken for them. Difficulty may sometimes be experienced in separating arthritis deformans, but the antecedent family and personal history and the habits of the patient, together with the absence of chalky deposits, serve to distinguish the one from the other disease.

Prognosis.—The likelihood of permanent cure is not encouraging, but by observing proper hygienic and dietetic measures, the attacks may be rendered infrequent and mild, and the development of severe internal lesions may be postponed—whereby the gouty subject may live the allotted time of man. The outlook, however, is bad when gout develops in a young subject under thirty years, and when arteriosclerosis and renal changes are advanced.

Treatment.—Attention to hygienic regulations is important. The patient should have plenty of fresh air; he should exercise regularly, but not too violently or sufficiently to cause exhaustion, since this may determine an attack of acute gout, besides being otherwise objectionable. He should bathe frequently—a morning cold bath or an evening warm bath, as best suits the individual patient. An occasional Turkish bath, as well as temporary sojourns at certain spas, are often very beneficial. The patient should drink freely of water—to the insistence upon which the high repute of certain "cures" is largely due. The drinking of the water itself, promoting free diuresis, rather than its alkaline contents, constitutes the useful part of the treatment. The waters that enjoy the greatest vogue are Saratoga, Bedford, and White Sulphur, in this country; Carlsbad, Homburg, Ems, Kissingen, in Germany; Aix-les-Bains,

Contrexeville, and Vichy, in France; and Buxton and Bath, in England. Sir William Roberts believes that the alkalescence of these waters is without influence on sodium biurate, and that the sodium salts are especially harmful; but since the drinking of large quantities of water in the solitude of one's room is a weariness to the flesh, while the same process at the spas not only requires considerable pecuniary outlay, but also promotes a healthy state of the mind, largely on account of the "accessories," such as regulated life, restricted diet, controlled exercise, baths, etc., the spas will continue to enjoy a well-merited repute.

Since most gouty subjects eat too much, the diet must be restricted, but the matter of regulation of the diet is entirely individual. In general the meats should be reduced, and the white meats should be preferred to the red, though the red meats need not be prohibited. Milk and eggs are usually well borne and may be given in large amounts. Green vegetables, such as lettuce, spinach, cauliflower, Brussel sprouts, asparagus, etc., are quite permissible. Though opinions differ with reference to carbohydrates and fats, they may be given in moderate amount. Ebstein favors the use of considerable butter, even in obese subjects, stating that not only do they improve in general condition, but actually lose some fat. Highly seasoned food, pastry, cheese, hot cakes and bread, tea, coffee, as well as liver, kidney, brain, sweetbread (rich in nuclear elements), and beef extracts (rich in derivatives of the xanthin group) must be prohibited. Sir William Roberts advises restriction of the sodium chloride of the food, believing that it favors the crystallization out of sodium biurate. In general alcohol must be strictly interdicted, especially the fermented beverages, such as beer, porter, ale, sweet and sour wines, champagne, etc. When really indicated, a small amount of whiskey or brandy, well diluted, may be permitted.

During the acute attack the patient should remain at rest, preferably in bed, and the diet should be restricted to a minimum of milk, eggs, and broths. The affected member should be enveloped in cotton wool or hot fomentations, or a 30 to 50 per cent. ichthyol ointment, or a saturated solution of magnesium sulphate, and then elevated. A blue mass pill or a purgative dose of calomel should be administered at the outset. Custom sanctions the use of the wine or the tincture of colchicum in 20 to 30 minim (1.25 to 2.0 c.c.) doses, combined with potassium acetate or citrate, 30 to 60 grains (2 to 4 grams), every four hours, until the acute symptoms subside. The colchicum should be stopped immediately improvement is effected. Colchicine, $\frac{1}{100}$ grain (0.0006 gram) or more, every two or three hours, has recently been recommended as a substitute for the wine or the tincture. In some cases morphine may be necessary to relieve the pain.

In the subacute and irregular forms of gout the salicylates have long enjoyed a widespread reputation and undoubtedly do good in many cases. In chronic cases, in addition to the foregoing measures, the use of potassium iodide, piperazin, guaiacum, quinine, and the bitter tonics, is sometimes attended by good results. The extract of thymus has been recommended.

OBESITY.

Obesity is a disorder of metabolism characterized by the excessive deposition of fat in the tissues.

Etiology.—Ordinary obesity is more common in women (especially Hebrews) than in men, and develops usually during the fifth decade (at or about the time of the menopause in women). In some cases there is an hereditary predisposition, in which event the obesity may develop during infancy, childhood, adolescence, or adult life. In the majority of cases the disorder is due directly to a sedentary life, indolence, deficient exercise, overeating, or excessive indulgence in alcohol; but especially to these combined. Many diseases that prevent adequate exercise, such as gout, chronic heart or lung disease, etc., or interfere with oxidation, such as anemia, etc., may lead indirectly to obesity.

Pathology.—The lesions consist in excessive, sometimes enormous, deposition of fat, not only in regions where fat is found normally, as the subcutaneous tissues, mesentery, about the kidneys, etc., but also in organs normally almost, if not quite, free from fat, especially the heart, the liver, etc. The fatty accumulation results from imperfect metabolism in general—perhaps lessened vitality of the cells in general or lessened requirement for food to form sufficient heat and energy. The fat is derived from the fat and the carbohydrate (starchs and sugars) of the food—that amount ingested in excess of the body needs being thus stored up; the non-nitrogenous moiety of the proteins imperfectly oxidized also goes to the formation of fat.

Symptoms.—The obvious manifestation is the excessive accumulation of fat in general, but especially in the chin, the back of the neck, the abdomen, the hips, etc. For a time, at least, there may be no symptoms aside from those due directly to the great bulk of tissue, and inconvenience in moving it about (walking, etc.). Sooner or later, however, dyspnœa, especially on exertion, muscular asthenia, and other evidences of embarrassed cardiac action (pulmonary congestion, bronchitis, cyanosis, œdema, oliguria, etc.) develop, and the physical signs of fatty heart may be elicited. Digestion may become impaired, constipation is common; the liver is often enlarged; amenorrhœa, sterility, and other disorders may occur. Arteriosclerosis, gout, diabetes, etc., are often associated, and there is a special predilection to secondary infection (lessened resistance to bacterial infection).

Diagnosis.—The diagnosis is easy, as a rule; myxœdema should be readily excluded. Other types of fatty accumulations sometimes must be taken into consideration, such as. (1) Adiposis dolorosa (Dercum), a disorder most common in middle life, in which, usually associated with more or less general obesity, irregular, symmetrical, painful, fatty masses, 1 to 3 or 4 inches in diameter (symmetrical lipomatosis), develop on the trunk and extremities (but not the hands, feet, or face). Pain may precede the development of the fatty

masses; and there may be paresthesias, anesthesias, and hyperesthesias. Asthenia, mental apathy, and psychoses may ensue. The disease is progressive, though of long duration, and not especially amenable to treatment. (2) Adiposis tuberosa simplex (Anders), a disorder resembling adiposis dolorosa, characterized by the development in the abdominal fat of fatty growths, sometimes painful to pressure. Unlike adiposis dolorosa, they are amenable to the ordinary dietetic regulations of obesity in general. (3) Adiposis cerebralis (dystrophia adiposo genitalis, Fröhlich), a condition of marked obesity developing in the course of tumor of the brain (especially the pituitary body, pineal gland), and associated with defective development of the genital organs. (4) Multiple lipomatosis—multiple symmetrical fatty tumors occurring in association with divers diseases of the nervous system (tabes, general paresis, etc.). (5) Symmetrical adenolipomatosis (MacCormac), a condition characterized by the development of fatty growths (containing sclerosed lymph nodes) in the neck, axilla, etc., and often associated with asthenia, emaciation, rapid pulse, enlarged spleen, etc. The nature of these disorders is not well understood. The lesions found have consisted of interstitial neuritis in the fatty masses, atrophy and compensatory hyperplasia of the thyroid, tumors of the pituitary, lymphoid hyperplasia, hypoplasia or retrogression of the sexual organs, and divers diseases of the central nervous system. Perhaps the cases are much related (disorders of internal secretion).

Prognosis.—The prognosis depends upon the nature of the underlying factors and the amenability of the subject to dietetic and other regulations.

Treatment.—The essence of successful treatment consists in bringing the diet within the requirements of the body as regards heat and energy, and in promoting oxidation and the destruction of fat by carefully regulated and graduated exercise, massage, and baths—all of which must be carefully supervised on account of the frequency of associated cardiac weakness. There are several systems of dietetic regulation, of which the best known are Ebstein's, Oertel's, von Noorden's, and Banting's (for details of which one may refer to special treatises). In general, however, the amount of food should be reduced, especially the starches and sugars; the fluid intake should be greatly restricted, and the small amount (2 to 3 pints daily) allowed should be taken preferably two hours after meals than with food; alcohol should be prohibited in most cases. Care should be taken to see that the nitrogen output does not exceed the intake, since this (destruction of the body protein) is answerable for most of the weakness that attends many "cures." Medicinal treatment is of secondary importance; a weak heart sometimes has to be stimulated, general weakness suggests the use of tonics, anemia requires the use of iron, etc. Thyroid extract is sometimes of service, but it must be employed with caution, since it leads to breaking down of the body protein as well as fat, and may induce thyroidism in those ill able to withstand its effects.

RICKETS.

(*Rachitis.*)

Rickets is a disease of infancy characterized by defective calcification of the growing bones and general disturbances of nutrition.

Etiology.—Rickets occurs in the great majority of cases before the end of the second year of life; it is uncommon before the sixth month, although cases in the newborn have been described (more likely achondroplasia); it is said rarely to develop about the time of puberty, rachitis tarda (rather osteomalacia of puberty). It is almost limited to the children of the poor and those who live amidst general unhygienic conditions; but it may occur under the best hygienic circumstances. Parental syphilis or tuberculosis, poor nutrition of the mother, and rapidly succeeding pregnancies, may be etiological factors. A faulty is diet the prime causative factor, whence the disease occurs in children fed on condensed milk, cow's milk, and proprietary foods, as well as in those nursed by the mother too long or when she has again become pregnant. Excess of carbohydrates (deficiency of fats and animal proteins) is believed to be the important factor.

Pathology.—The chief lesions occur in the bones, and consist of hyperemia of the cartilage, bone marrow, periosteum, and bony tissue itself—which results in abnormal development of the growing bone, as well as changes in the bone already formed. In the long bones, ossification of the cartilage at the junction of the epiphysis and diaphysis is delayed and irregular; the outer, bluish, proliferative cartilaginous area exhibits irregular multiplication of the cells and a soft fibroid matrix invaded by bloodvessels; the inner yellowish ossifying layer becomes irregularly thickened and shows imperfectly calcified areas; the two layers are no longer sharply defined, but gradually merge. The osteoblastic layer of the periosteum of the shaft of the bone becomes thickened and nodular, the medullary cavity, continuously enlarging, results in lessening of the thickness of the osseous shaft. In consequence, then, of delayed and irregular ossification (less than one-half of the normal amount of lime salts may be present) the ends of the long bones are softened and nodularly enlarged and the shafts irregularly thickened—whence they readily become bent, distorted, or fractured. Ultimately they become ossified and hard, but they are likely to remain deformed. The liver, spleen, and mesenteric lymph nodes may become enlarged. The nature of the process is not well understood; it is not due to deficiency of lime salts in the food, nor to bacterial infection.

Symptoms.—The onset of the disease is insidious. The early symptoms, which usually develop about the time of dentition, consist, as a rule, of general disturbances rather than obtrusive changes in the bones, so that, especially among the well-to-do, the disease may be overlooked for some time. The child is nervous, restless, irritable, sleeps poorly, and may have attacks of fever; he cries when touched, seems to be

generally hyperesthetic (bony prominences and soft parts), and throws off the bedclothes; he does not thrive, he sweats considerably, especially about the head and neck when asleep; dentition is delayed or irregular; the development of the speech function is retarded; gastrointestinal disturbances, such as poor appetite, vomiting, flatulence, and diarrhœa, are common; emaciation and anemia supervene, the musculature becomes soft and flabby, and the child is unable to sit erect or walk.

In the course of time characteristic changes appear in the bones. The skull is relatively large, the sutures and the fontanelles remain unclosed (even in the third and fourth years), and the forehead is high, wide, and prominent; in young infants the bones are soft and thin (craniotabes) and yield readily to pressure. A systolic murmur may be audible over the open anterior fontanelle. The costochondral junctions being swollen occasion the characteristic bending of the ribs (rachitic rosary), which usually disappears by the fourth to the sixth year; atmospheric pressure on the softened ribs (especially if associated with difficulty in breathing) may give rise to a groove along the costochondral junction, which when associated with a prominent sternum gives rise to the so-called pigeon breast or chicken breast or keel-shaped thorax; there may also be a groove (Harrison's) along the attachment of the diaphragm. The lower ends of the radius and ulna (junction of the epiphysis and diaphysis) become thickened, enlarged, and deformed. Similar changes may occur in the clavicle, the tibia (especially the lower end), and the other bones of the arms and legs, in the vertebræ (kyphosis, scoliosis, lordosis), and in the pelvis (rickety pelvis; subsequent difficulties in parturition). The changes in the bones of the legs (occasioning pain) and the associated muscular weakness lead to delay in walking, and may simulate paralysis (pseudoparalysis of rickets); if the child does walk, bow-leg or knock-knee is an almost inevitable result. Greenstick fractures of the long bones are common.

In addition, rickety children are often the subject of night terrors (pavor nocturnus), convulsions, tetany, laryngismus stridulus, enuresis, etc. The liver and spleen are often enlarged—which together with the marked intestinal disturbance (flatulence and muscular atony) gives rise to the characteristic "pot-belly." Bronchopneumonia and other pulmonary complications are common.

Diagnosis.—The characteristic changes in the bones make the diagnosis easy; but special efforts should be made to recognize the disease early: open fontanelle, craniotabes, head sweating, throwing off of the bedclothes, delayed dentition, and crying when touched, are important diagnostically. Infantile scurvy (misnamed acute rickets) may be excluded by the aforementioned signs and symptoms, especially the changes in the cranium, and by the absence of the characteristic scorbutic changes in the gums and the bones of the legs. Syphilis may be excluded by the absence of other, especially cutaneous and mucous membrane, evidences of syphilis, and should the epiphysis be involved, by the absence

of tender, painful doughy swellings, and perhaps dissociation of the epiphyses and diaphyses.

Prognosis.—The disease is chronic in course, but tends to spontaneous recovery within a year or more; under proper treatment cure may be effected within several months. Deaths are usually due to complications, especially secondary infections.

Treatment.—The best hygienic conditions should be procured—fresh air, sunlight, suitable clothing, frequent bathing, cool breezes, low temperature during the summer, etc. If the child is young, and the mother cannot satisfactorily nurse it, or if she is ill or pregnant, a wet nurse may be procured. Otherwise proper attention must be paid to the diet; cow's milk properly modified to suit the age and digestive abilities of the child is the best; barley water, oatmeal gruel, albumin water, and beef juice may be added. Phosphorus is the best remedy; it leads to calcium retention in rachitis, and exerts a specific effect on rachitic bones, increasing their calcium content; $\frac{1}{100}$ grain (0.0006 gram) thrice daily may be given in olive oil or cod-liver oil; and cod-liver oil may be rubbed into the skin. The hypophosphites, arsenic, syrup of iron iodide, and other iron preparations also are of service, especially in anemic cases. The child should not be allowed to walk until the condition is cured.

SCURVY.

(*Scorbutus.*)

Scurvy is a chronic disorder of nutrition associated with dietetic errors (deprivation of fresh food), and characterized by weakness, anemia, spongy gums, and hemorrhages.

Etiology.—Formerly the disease was prevalent among sailors, soldiers, miners, prisoners, and others deprived for a long time of fresh food; at present it is rare, but occasionally seen in seaports, in famine-stricken districts, and among the poor. Epidemics have been observed. Unhygienic conditions, debilitating influences, exposure to wet and cold are believed to be predisposing factors; but the chief factor is in some way connected with the prolonged lack of fresh food, especially of fresh meat and vegetables; that is, it follows a diet consisting largely of dried or salt meat, dried vegetables, cereals, etc. Infantile scurvy (Barlow's disease, incorrectly called acute rickets) occurs among the well-to-do as well as among the poor, but in artificially fed infants, those fed on condensed milk, sterilized milk, proprietary food, etc. The ultimate etiological factor has not been determined; it is believed to be due to deficiency of potassium salts, especially the citrate, but also the tartrate and maltate (Buzzard); and to diminished alkalinity of the blood from an insufficient supply of vegetable salts (Rolfe). Recently Wright has contended that the condition is an acid intoxication due to an excess of foodstuffs yielding an acid ash after incineration. These, which he calls scorbutic, are oats, barley, fresh meats, salt meats, ship biscuit, wheat, eggs, rice, maize, peas, and beans; the neutral or non-scorbutic

foodstuffs comprise sugar, vegetable oils, and animal fat; and the alkaline or antiscorbutic foodstuffs comprise carrots, turnips, potatoes, onions, milk, blood or meat containing blood, lime juice, orange juice, wood, and green herbage. Holst contends that the disease is not an acid intoxication, but is due to the lack in the food of some unidentified but easily decomposable constituents (perhaps of the nature of an enzyme) —present in antiscorbutic foods, but lacking in cereals (flour, grains), because they are dry.

Pathology.—The chief lesions are hemorrhagic in nature: ecchymoses in and beneath the skin and mucous (sometimes also the serous) membranes; larger intermuscular and sometimes intra-articular hemorrhages; subperiosteal hemorrhages (most common about the femur, but also elsewhere, as beneath the alveolar periosteum, etc.); and hemorrhages into the epiphyseo-diaphyseal junction of the long bones, associated with the deposition of fibrillated or reticular tissue which may soften and lead to separation of the epiphyses and diaphyses (which, however, may be retained in position by the periosteum). There may be some absorption of lime salts or decalcification (acidosis) and consequent fragility and ready fracturing. The gums become swollen, spongy, hemorrhagic, and sometimes ulcerated, and the teeth may become loosened. Ulcerations may develop in the ileum and colon. The spleen may be enlarged and the liver, kidneys, and heart reveal the usual results of marked anemia (parenchymatous and fatty degeneration).

Symptoms.—The onset is insidious, the early symptoms consisting usually of general ill health, weakness, anemia, and loss of flesh. The hemorrhagic manifestations usually begin in the gums, which (often first about the canine teeth) become soft, swollen, spongy, hemorrhagic, and perhaps ulcerated; the teeth may fall out; the breath is very offensive, and the adjacent lymph nodes become swollen. Petechiæ and ecchymoses develop in and beneath the skin, especially of the legs; larger hemorrhages may develop in and between the muscles, especially of the calves, thighs, buttocks, abdominal wall, shoulder, etc. (scurvy induration or sclerosis); or beneath the periosteum, especially of the femur (painful nodular protuberances). Epistaxis, gastro-intestinal hemorrhage, hematuria, and other mucous membrane bleedings, and serous membrane hemorrhages (especially into the larger joints) may occur; parenchymatous (visceral) hemorrhages are rare. The hemorrhagic effusions, as a rule, become absorbed, but may break down into foul ulcerations. There may be erythematous, urticarial, and other skin eruptions. Cardiovascular and pulmonary symptoms of anemia (dyspnœa, palpitation of the heart, œdema, etc.); headache, mental depression and apathy, or delirium, etc., may supervene. The total acidity of the gastric juice is usually lessened, and the free HCl may be entirely absent. Necrosis, fracture, epiphyseal separation of the bones (femur, clavicle, ribs) may occur. Usually there is constipation, but there may be diarrhœa (scorbutic dysentery). The temperature is usually normal or subnormal (in the absence of complications).

Diagnosis.—The diagnosis is easy from a knowledge of the etiological factors (especially in the event of epidemics), the gingival sponginess and hemorrhage, deep-seated intramuscular and subperiosteal hemorrhages, especially in the legs, and speedy cure under the influence of an antiscorbutic diet. These serve to exclude purpura and hemophilia.

Infantile scurvy (Barlow's disease, formerly incorrectly described as acute rickets) is quite the same as the disease in the adult. In the beginning there is usually at least some slight wasting or obviously impaired nutrition, apathy, disinclination to movement, and general irritability. Soon the child is observed to cry when disturbed or touched (especially the lower limbs); pseudoparalysis of the legs seems to have developed, and an irregular, painful, and tender swelling of one or both thighs, beginning above the epiphyses, becomes apparent (subperiosteal hemorrhage). Similar phenomena may develop elsewhere—in the vertebræ, the scapulæ, the bones of the arms, etc., and the process shows a tendency to become symmetrical. The epiphyses may become separated from the shafts of the long bones (femur, costal cartilages, etc.), and crepitus may be elicited. Swelling of the gums (if the child has teeth), and other mucous membrane, cutaneous, and intermuscular hemorrhages may supervene; hematuria may be the only noteworthy symptom. Œdema of the eyelids associated with ecchymosis of the lids or conjunctiva is not infrequent. Asthenia is usually marked; anemia and pallor are moderate; there may be slight fever (101° to 102°). Rickets may be excluded by the tender painful swellings of the shafts of the bones, especially of the legs, and the sponginess and hemorrhage of the gums. Rickets is sometimes believed to be associated with scurvy (scurvy-rickets); perhaps it predisposes to scurvy.

Prognosis.—The prognosis is good if the disease is recognized and the proper treatment is instituted. In neglected cases death may ensue from hemorrhage, asthenia, anemia, secondary infections, etc.

Treatment.—Nowadays the supplying of sailors, soldiers, and others with a suitable antiscorbutic diet effectually prevents the disease. When the disease has developed, a generous supply of fresh meat, fresh vegetables, and the juice of two or three lemons, oranges, or limes daily (sweetened if desired) commonly lead to cure. If the gums are much swollen the diet should be liquid or semisolid for a time; the gums should be painted with silver nitrate (2 to 5 per cent.); and boric acid (3 per cent.), or the solutions mentioned on page 51, should be used as a mouth wash. Hydrochloric acid, stomachics, bitter tonics, strychnine, alcohol, iron, etc., are sometimes required for obvious indications. Children should be given mother's milk if possible, or fresh unsterilized cow's milk suitably modified, and orange juice or lemon juice, other fruit juices, and meat juice, perhaps with mashed potatoes and vegetable purees. Iron, arsenic, cod-liver oil, brandy, etc., are useful adjuvants. In the event of separation of the epiphyses, splints should be applied.

AMYLOIDOSIS.

(*Amyloid Disease; Lardaceous Disease; Waxy or Bacony Degeneration.*)

Amyloidosis is a disorder of metabolism that develops in the course of prolonged suppuration, especially tuberculous or syphilitic disease of the bones, and is characterized by the formation in the tissues and organs of lardacein, a combination of chondroitin-sulphuric acid and a protein.

Etiology.—Amyloidosis follows prolonged suppuration and long-continued loss of the body proteins. It is most common in advanced tuberculosis, especially of the bones and joints (Pott's disease of the vertebræ, hip disease, etc.); it occurs also in advanced syphilis, sometimes even in the absence of suppuration; in non-tuberculous and non-syphilitic suppurative processes, such as osteomyelitis, empyema, etc.; in non-suppurative disorders leading to anemia and cachexia, such as malaria, leukemia, carcinoma, chronic nephritis, prolonged lactation, etc.; and sometimes it occurs without obvious cause. The disorder is most common in young adults (before the fortieth year), and in males, since they are more subject than females to the initiating causes.

Pathology.—The lesions are sometimes confined to a single organ; usually they are widespread, being most common in the kidneys, spleen, and liver (which often are involved concurrently); they occur also in the small intestine, the lymph nodes, the adrenals, the stomach, the large intestine, the heart, the pancreas, the thyroid, the bone-marrow, etc. The solid viscera become enlarged, firm, dense, and heavy; they take on a homogenous, semitranslucent or wax-like appearance, and give a characteristic reaction with iodine and other substances. The lardacein is not, as was formerly supposed, an infiltration derived from the blood, but is a formation developed *in situ* in consequence of an interaction between chondroitin-sulphuric acid brought by the blood or the lymphatics and the local protein; the process is in some way determined or influenced by bacterial (and perhaps other) toxins, notably the toxins of staphylococci, streptococci, etc. The suppurative processes in which amyloidosis occurs are in most instances examples of secondary pyococcic infections. The amyloid material is deposited in the outer surface of the capillary vessels, and in the intima and media of the arterioles; the adventitia is rarely, and the endothelium never, affected. In the kidneys the Malpighian tufts are especially involved, in the liver the midzonal region of the lobules, in the spleen the Malpighian bodies, and in the mucous membranes the arterioles.

Symptoms.—The symptoms are largely those of the antecedent disorder, of which the amyloidosis is the end result. With gradual increase in the anemia, cachexia, and weakness, the abdomen is observed to enlarge, or polyuria, diarrhœa, or œdema may develop. The abdominal enlargement is readily found to be due to enlargement of the liver or spleen or both; the liver may become enormous, and is smooth, hard, firm, and has a somewhat sharp or rounded edge; the spleen is enlarged

and presents a firm, resisting, blunt edge. There is rarely any jaundice, and ascites develops only as part of anasarca. Involvement of the kidney is shown in œdema and increase in the amount of the urine, which is pale in color and of very low specific gravity; it contains usually a considerable amount (sometimes very little) albumin, and hyaline and waxy casts (which may give the amyloid reaction). In the event of associated nephritis (which is common) cellular casts, arterial hypertension, arteriosclerosis, hypertrophy of the heart, retinal changes, etc., may be found. Involvement of the intestine may be symptomless, but if advanced, the lesions are likely to be disclosed by more or less intractable and painless diarrhœa. Changes in the cutaneous vessels, and a failing heart, are often in part answerable for the œdema.

Diagnosis.—The diagnosis is readily made, the combination of the etiological factor, progressive increase of anemia, cachexia, and weakness, enlargement of the liver and the spleen polyuria, albuminuria, and diarrhœa being unmistakable.

Prognosis.—The prognosis depends chiefly on the initiating cause; the advent of the amyloidosis only makes the prognosis hopeless.

Treatment.—The treatment is wholly that of the primary disorder.

INFANTILISM.

Infantilism is a condition characterized by the persistence beyond the ordinary age of puberty of the physical and mental attributes of childhood, including hypoplasia of the sexual organs and absence or abeyance of the secondary sexual characteristics. The individual remains a child in body and in mind (emotions and intelligence), the adult bodily characteristics remain in abeyance or become ill developed, the union of the epiphyses and diaphyses of the long bones fails or is much delayed. In the male, the facial, axillary, and pubic hair does not grow, nor does the larynx enlarge or the voice deepen; in the female, the axillary and pubic hair does not grow, the breasts do not develop, the hips do not widen, and there is little indication of the female waist. The underlying causes may be present at birth (congenital cases); or the condition may be due to factors that become operative in early or late childhood. The ensuing phenomena vary accordingly: in some the infantilism is complete, in some incomplete, whereas in other, perhaps related, cases the onset of puberty is merely delayed—full and normal development ultimately ensuing. Several types may be distinguished:

The Lorain Type.—In this, beyond the ordinary age of puberty, the figure remains small and childlike as regards height, but the general contour is that of approaching puberty. Although there is no growth of the facial, pubic, or axillary hair, nor of the breasts in the female, the genital organs are well formed, although small; the head is relatively small, the shoulders are fairly broad, and the bony prominences and muscles are distinct—a miniature man or woman who has retained the characteristics

(mental as well as physical) of childhood. The cause of the disease is not known: indeed, it is sometimes referred to as idiopathic; but syphilis, alcoholism, and consanguinity in the parents are believed to be of etiological importance; and defective development of the cardiovascular system is believed to be the main factor.

The Cardiac Type.—Congenital heart disease, and also heart disease beginning early in life, especially pulmonary or aortic stenosis, may lead to infantilism. In some otherwise fairly normal, although small adult individuals a concealed or latent mitral stenosis may be found.

The Metabolic Type.—Disorders of internal secretion apparently may give rise to several varieties of infantilism. Thyroid inadequacy, congenital or developing early in life, occasions a so-called myxœdematous infantilism or cretinism (Brissaud's type; Hertoghe's hypothyroidism), which may be cured by thyroid extract. Another form occurs in connection with disease of the pituitary: hyperpituitarism originating in youth gives rise to gigantism, which may be associated with infantile proportion of the body, infantile genital organs, delayed union of the epiphyses and diaphyses, etc. Hypopituitarism originating in youth gives probably rise to infantilism of mind as well as body. Gilford's cases of dwarfs, micromegaly, ateliosis (continuous youth and premature old age), or progeria perhaps belong to this category. A pancreatic form, supposedly due to deficient pancreatic secretion and associated with recurring attacks of severe diarrhœa in children, has been described by Byron Bramwell. Improvement has followed the use of pancreatic preparations.

The Intestinal Type.—Herter describes as *intestinal infantilism* a condition occurring in childhood, and characterized by arrest in the development of the body (skeleton, muscles, and organs), maintenance of good mental powers and fair development of the brain, marked abdominal distention, slight, moderate, or considerable degree of simple anemia, the rapid onset of physical and mental fatigue, irregularities of intestinal digestion resulting in frequent diarrhœal discharges, excessive appetite, various minor signs of nervous instability, subnormal temperature, cold hands and feet, and slight signs of rickets. Indol and phenol compounds occur in the urine; fat absorption is impaired; and the resorption of calcium and magnesium is deficient. Herter attributes the condition to the overgrowth and persistence of the bacterial flora of the intestine belonging normally to the nursling period (Bacillus bifidus and Bacillus infantilis). The condition is persistent, and relapses are common; some of the children die of acute intestinal infections; others are likely to be permanently dwarfed; but normal growth may follow careful dietetic regulations.

SECTION IV.

DISEASES OF THE DUCTLESS GLANDS AND OF INTERNAL SECRETION.

Pathological Physiology.—In addition to the several organs, such as the stomach, the liver, the pancreas, the kidneys, the testicle, etc., which produce a secretion that can be readily procured and studied, other organs produce a secretion not so open to investigation, a secretion which, since it is not discharged through the medium of a duct from the organ that produces it, is spoken of as an internal secretion, and the organ in question as a ductless gland. In a certain sense all of the body cells produce an internal secretion—the result of their metabolic activity—which they give off into the lymph or blood stream. Some of these are the end results of destructive metabolism and of no further use in the economy—indeed, they may be distinctly harmful if not excreted (certain forms of auto-intoxication); others are more or less essential to the orderly performance of the functions of other organs, necessary to the preservation of life and the maintenance of health; these are the internal secretions in a restricted sense, and the "glands" that produce them are the "ductless glands" or the glands with an internal secretion. They comprise especially the thyroid, the parathyroids, the adrenals (and the chromaffin system), and the pituitary body, which are endowed with specific functions that will be discussed under the respective headings; perhaps the carotid and coccygeal glands also should be here included, but nothing is known of their function. But other organs produce internal secretions: some of these also are "external secretion;" thus, the pancreas pours into the duodenum a secretion that contains trypsinogen, which hydrolyzes protein, diastase (amylase), which hydrolyzes carbohydrates, lipase (steapsin), which saponifies and indirectly emulsifies fats, and perhaps other enzymes (rennin, lactase, etc.); but, in addition, the islands of Langerhans of the pancreas, which are not in communication with the pancreatic duct, produce an internal secretion that finds its way directly into the blood and controls the katabolism of the dextrose of the blood, muscles, and other tissues—a subject discussed under diabetes. The liver and the kidneys, in a certain sense, also have an internal secretion in addition to the more obvious external secretion—

the bile and the urine. The liver, by a process that may be described as internal secretion, produces glycogen and an enzyme (urease) that leads to formation of urea from protein derivatives; and the kidneys appear also to produce a proteolytic enzyme subserving the same purpose, though probably acting in the liver or the muscles rather than in the kidney itself. The testicle and the ovaries, in addition to their specific secretions which are discharged externally, produce also an internal secretion which is credited with the production of the secondary male and female characteristics respectively, and seems to exercise some tonic influence on the neuromuscular system. Castration in either sex results in lessened oxidation, accumulation of fat, and in more or less loss of the secondary sexual characteristics, which loss may be prevented or retarded by the use of testicular or ovarian preparations respectively. The duodenum produces secretin which stimulates the pancreas to activity and enterokinase which activates the proteolytic ferment of the pancreatic juice (trypsinogen); but these can scarcely be called internal secretions in a restricted sense. Organic substances subserving such a stimulating purpose have recently been spoken of as kinases, while similar inorganic substances have been called activators. The spleen, the lymph nodes, the hemolymph nodes, and the thymus also, perhaps, produce an internal secretion, but there is no conclusive evidence of the fact; their diseases are discussed under diseases of the blood and hemopoietic system.

The ductless glands, or the glands with an internal secretion in a restricted sense, are subject to many complicated and ill-understood relationships; thus, in diseased states at least, there is a more or less marked relationship between the thyroid and the pituitary, between the thyroid and the thymus, between the pituitary and the testicles and the ovaries, and between the thyroid, the pancreas, and the adrenals. The functional harmony of these organs is maintained and their correlated interaction is effected through the medium of chemical substances, so-called hormones, that circulate in the blood and other fluids of the body. Whereas, the pancreas and the thyroid, and the pancreas and the adrenals (and the chromaffin system) each inhibits the metabolic activities of the other, the thyroid and the adrenals (and the chromaffin system) seem the one to augment the activities of the other. Experimental removal of one of these glands not only results directly in a loss of its functional activities, but leads also indirectly, since restraint is removed, to an increased or diminished functional activity of the correspondingly related gland; and augmented functional activity (such as may be attained by feeding experiments or the injection of active principles or extracts) has a contrary effect. Thus, for instance, loss or diminution of the internal secretion of the pancreas, in consequence of the corresponding loss of inhibition, is associated with hyperfunction of the thyroid (increase of protein metabolism) and of the adrenals and the chromaffin system (adrenal glycosuria). Similar phenomena occur also in human subjects: thus, hyperthyroidism is sometimes associated with alimentary glycosuria

or diabetes. The pigmentation of Graves' disease (resembling that of Addison's disease) suggests a relationship between the thyroid and the adrenals. There are many other evidences of mutual inter-relationship between the ductless glands, which, however, are ill understood.

DISEASES OF THE THYROID GLAND.

Pathological Physiology.—The normal physiology of the thyroid is ill understood. In consequence of the activity of its specific principle iodothyrin (a constituent of colloid and not found elsewhere in the body), and perhaps also of other unknown principles, it exercises a profound influence on metabolism. It detoxifies intermediate products of metabolism or gives over into the circulation an internal secretion essential to the normal functional activity of other organs, acting, therefore, as a hormone and seeming to have a specific action on the central and the sympathetic nervous systems and on the skin and the subcutaneous tissues. Injection of thyroid preparations increases protein and fat katabolism. An influence of deficiency of secretion on carbohydrate metabolism is evident in the fact that after thyroidectomy injections of adrenalin do not cause glycosuria; that is, the limits of carbohydrate metabolism are increased. Deficiency of secretion also exercises a restraining influence on the healing of fractures; an excess seems to accelerate the healing. Destruction or loss of function of the thyroid (athyrea, hypothyroidism) results in congenital or acquired myxœdema; and an excess of function (hyperthyroidism), or perversion of function (dysthyreosis), is the basis of Graves' disease; whence the two diseases represent the one the antithesis of the other. In the one the gland is atrophic or absent, in the other hyperplastic and hyperactive; in the one metabolism is deficient and the patient increases in weight, in the other metabolism (especially the consumption of oxygen) is augmented and the patient usually emaciates; in the one the patient is dull, apathetic, and mentally deficient, in the other nervous and highly excitable; in the one the skin is dry, thick, cool, and inactive, in the other moist, warm, and active (erythema, flushing, sweating); in the one the temperature is low, the pulse is slow, and the urine lessened, in the other the temperature is increased, the pulse is rapid (tachydcardia), and the urine increased; in the one (which may result from excision of the thyroid) the symptoms may be ameliorated or the condition entirely cured by the use of thyroid extract, in the other, symptoms are made worse by thyroid extract—which may also provoke similar phenomena in normal individuals—and the condition may be benefited or cured by partial thyroidectomy.

Thyroid preparations have been used with some success in certain disorders of metabolism, such as obesity, arthritis deformans, etc. Their use, in obesity, for instance, in which they seem especially indicated to augment the katabolism of fat, is not without danger, since although they have a much greater accelerative influence on protein katabolism,

they may cause glycosuria or diabetes (which is often latent in obese subjects), and a fatty heart may not be able well to maintain the provoked increased activity.

ACUTE THYROIDITIS.

Etiology.—Acute inflammation of the thyroid occasionally occurs, usually in young subjects, in consequence of trauma or of bacterial infection. The infectious cases are rarely primary; usually they are secondary, occurring in the course of typhoid fever, scarlatina, diphtheria, measles, influenza, smallpox, some cases of recent syphilis, etc. The lesions may be simple or purulent.

Symptoms.—The symptoms consist of local pain and tenderness, sensation of fulness in the neck, perhaps difficulty in swallowing, and an enlarged and more or less tender thyroid. In the event of suppuration a local area of softening or fluctuation may be detected. The general symptoms are those of the primary infection, augmented perhaps by the local inflammation.

Diagnosis.—In the diagnosis one must exclude simple congestion, such as occurs especially in women from emotional excitement, disturbance of the sexual organs, interference with the circulation (tight collars), etc.

Prognosis.—The prognosis, as a rule, is good as regards the thyroiditis itself, but modifications in the gland may lead later to Graves' disease or myxœdema.

Treatment.—The treatment is that of the primary disorder, with local antiphlogistic measures (especially an icebag), and incision in the event of suppuration.

GOITRE.

(Simple Goitre; Struma; Bronchocele.)

Goitre is a term indiscriminately applied to any enlargement of the thyroid gland. It is often qualified by the terms benign and malignant (comprising the tumor formations); and a simple goitre is differentiated from the so-called exophthalmic goitre.

Etiology.—Simple goitre occurs as a sporadic, an endemic, or an epidemic disease. The sporadic disease occurs in all parts of the world. The disease is endemic especially in the Swiss and Italian Alps, but also in certain regions of France, Spain, Germany, England, Asia, and in our own country in the region of the great lakes; in these endemic regions small epidemics sometimes occur from time to time, as well as occasionally in barracks, schools, etc. The disorder is more common in women than in men (six or more to one). It is sometimes congenital. The goitre begins to develop usually at puberty or soon thereafter, and may continue to increase in size throughout life, although, attaining a considerable size by the time adult life is reached, it may remain stationary.

The cause of the disease is not known, although it has been attributed to the water, air, and soil of endemic centres; special significance has been attributed to the drinking-water—mineral, radio-active, and bacterial constituents.

Pathology.—The thyroid is more or less enlarged, either uniformly or one lobe more than the other; the shape of the organ is generally more or less preserved; the enlargement is often nodular, although it may be smooth. On section it resembles the normal gland, although often the colloid is obviously increased. Parenchymatous (or colloid), cystic, and vascular goitres are sometimes distinguished. Hyaline degeneration, calcareous infiltration, and fibrosis are not uncommon in advanced cases. MacCarthy differentiates the following types or stages of goitre: (1) Cystic goitre (usually spoken of as simple or colloid goitre), composed of many small and large cysts with colloid contents; it leads to enlargement, but no symptoms other than those due to pressure. (2) Hypertrophic parenchymatous goitre—hypertrophy of the parenchyma with little or no cyst formation. (3) Papillary cystic goitre—cyst formations with colloid contents, papillary ingrowths into the alveoli, or cysts with consequent increased secreting surface. Hyperthyroidism may occur in types 2 and 3. (4) Hypertrophic fœtal thyroid —a small amount of epithelial tissue and a great amount of interglandular connective tissue—the goitre of the cretin with tumor formation and hypothyroidism. (5) Fœtal adenoma or adenomatous hypertrophy of the fœtal thyroid, instead of connective tissue overgrowth; it may occur as an encapsulated tumor, occasioning inconvenience only by pressure, or degenerative or circulatory changes may occur and lead to hyperthyroidism.

Symptoms.—The chief symptom is the goitre itself, which may be very small or very large, and involve the entire gland, or one lobe only or disproportionately, or the isthmus; it may grow larger behind the sternum or the clavicles. When large it may lead to inconvenience on account of its size and weight, or it may cause compression phenomena —on the trachea, œsophagus, arteries, veins, and nerves of the neck. Sudden death occasionally occurs in large goitres from pressure on the vagus, hemorrhage and compression of the trachea, œdema of the glottis, etc.

Diagnosis.—The diagnosis, as a rule, is obvious; one must exclude tumors and exophthalmic goitre.

Prognosis.—The condition is essentially chronic, although some cases, so-called acute goitres, that develop in newcomers into an infected region, often disappear if the subject soon leaves. The prognosis is influenced by the size and direction of growth and the presence or absence of compression phenomena.

Treatment.—Boiling the drinking-water in goitrous regions is usually advised as a prophylactic measure. Change of residence to a non-goitrous region is much preferable. Counterirritants, iodine externally, and the iodides and ergot internally, and the *x*-rays locally are some-

times of service, especially if the goitre be small and of recent development. Large and growing goitres should be treated surgically—removed in part.

HYPERTHYROIDISM AND EXOPHTHALMIC GOITRE.

(Graves' Disease; Basedow's Disease; Parry's Disease; Flajani's Disease.)

Exophthalmic goitre is a condition of aggravated hyperthyroidism, characterized by enlargement of the thyroid, exophthalmos, tachycardia, muscular tremor, and vasomotor disturbances; it takes its name from the two obtrusive manifestations of the disorder, but there are many more or less ill-developed cases that present neither goitre nor exophthalmos and are preferably spoken of as hyperthyroidism.

Etiology.—From 80 to 95 per cent. of the cases occur in women, and most frequently between the ages of fifteen and thirty years. The disease is rare during childhood, but is not uncommon in the fourth decade. The cause is not known; it develops not uncommonly at or shortly after puberty; its onset is sometimes attributed to fright, fear, excitement, worry, anemia, chlorosis, gastro-intestinal disturbances, infections, etc. Perhaps these factors, if at all operative, are to be interpreted as furnishing nervous, toxic, infectious, or metabolic stimulation to the thyroid. Etiological significance is sometimes attributed to locality and climate, but the nature of the relationship, if any, is not understood.

Pathology.—The significant lesion consists of enlargement of the thyroid, which varies considerably in different cases; it may be very slight or very great, symmetrical or asymmetrical, firm or soft and elastic (parenchymatous, fibrous, or cystic). The characteristic microscopic lesions consist of changes in the size and configuration of the alveoli, due especially to hyperplasia and infolding of the epithelium; increase in the size of the epithelial cells—the normal low cuboidal cells giving way to high cylindrical cells; and gradual disappearance and absorption of the colloid material; with these there is also associated an increase in the bloodvessels and the connective tissue stroma. The symptoms are attributed to thyroid toxemia due to the absorption of the products of a hyperactive (perhaps perverted) thyroid (hyperthyroidism); this consists in part of iodothyrin, a constitutent of the colloid, which is usually lessened in the exophthalmic goitres; perhaps also to other unknown substances. Sometimes lesions are found in the central nervous system (especially the restiform bodies of the medulla), and in the sympathetic nervous system (especially the cervical sympathetic); but they are inconstant and probably not significant. The muscles, despite the tremor, show no noteworthy lesions aside from some atrophy, especially of the muscles of the neck, hand, arms, and peroneus; in advanced cases the heart becomes hypertrophied, dilated, and degenerated.

Symptoms.—The onset, as a rule, is insidious, the symptoms mild, moderate, or severe, and the course protracted—chronic cases; occa-

sionally the onset is abrupt—acute cases, which also may be mild, moderate, or severe; the severe cases may run a rapid course and soon lead to death. In some cases the symptoms of hyperthyroidism are present from the beginning (primary cases), whereas in others they supervene upon a goitre already, perhaps for a long time, existent (secondary cases). In some cases the symptoms are many and obtrusive (outspoken or complete cases), whereas in others they are few, irregular, atypical (incomplete, rudimentary cases, *formes frustes* of the French). Remissions and exacerbations are common.

The characteristic features of the disease consist of enlargement of the thyroid, exophthalmos and other ocular symptoms, tachycardia, muscular tremors, and various vasomotor disturbances. The goitre is almost invariably present, some enlargement being present in many cases in which it is not suspected; but there is no relationship between the size of the goitre and the severity of the symptoms; it is often present from the beginning, but sometimes it develops later, at least in an obtrusive form. It may be slight, moderate, or extreme, and vary in size from time to time (during menstruation, pregnancy, times of worry, etc.); it may be unilateral, bilateral, symmetrical or asymmetrical; firm or soft, elastic, and semifluctuating. It is rarely painful except when acutely enlarged (vascular enlargement), and unless very large gives rise to no noteworthy signs of compression of the trachea or adjacent organs. It may pulsate, and on ausculation reveal a systolic (arterial) and sometimes also a diastolic (venous) murmur.

Exophthalmos is present in from 75 to 90 per cent. of the cases, and although a very obtrusive symptom, is not, as a rule, an early symptom. It varies much in degree (25 to 30 millimeters, as measured by the Hertel exophthalmometer, the normal being about 20 millimeters in adults, and less in children); ordinarily it just about prevents complete closure of the eyelids, but rarely the eyeballs may be advanced altogether beyond the eyelids. Usually it is bilateral, although unequal; it may be almost if not completely unilateral, and perhaps associated with homolateral goitre. There are a number of associated ocular phenomena: A peculiar staring expression (widening of the palpebral fissure), the whites of the scleræ showing all around the cornea (Dalrymple's sign), associated with infrequent and incomplete reflex winking (Stellwag's sign); lagging of the upper eyelids in downward movement of the eyeballs—incoördination in the movements of the eyelids and the eyeballs (von Graefe's sign); lack of the power of binocular convergence unassociated with diplopia (Moebius' sign); difficulty or inability to evert the upper lid (Gifford's sign); tremor of the upper lid when it has been closed gently (Rosenbach's sign); sudden elevation of the upper lid, exposing the sclera, if the patient looking straight forward is directed quickly to look upward; that is, the lids contract spastically in advance of the elevating eyeball (Kocher's sign). All of these eye signs, including the exophthalmos, are due to toxic contraction of the unstriped orbital muscle arising from the orbital septum anteriorly and inserted just

posterior to the equator of the eyeball, and spasm of the unstriped superior and inferior tarsal muscles of Müller; vascular engorgement and œdema of the orbital cavity also have been looked upon as causes. There may also be absence of wrinkling of the forehead when the patient looks upward (Joffroy's sign); a sensation of tension or strain in or behind the eyeballs; tremor of the eyeballs; paralyses of the ocular muscles; excessive dryness or epiphoria; retinal, pupillary, or corneal (ulcerative) changes; pigmentation of the eyelids (Tellais' sign), etc.

Tachycardia is always present, and is commonly the earliest noteworthy manifestation. The pulse is usually above 100, and not infrequently 160 to 180 or more. There is associated palpitation and overaction of the heart, throbbing of the peripheral arteries, especially of the neck, a capillary (and perhaps a venous) pulse, early a functional systolic murmur, later evidences of hypertrophy and dilatation of the heart (especially of the left ventricle), with relative mitral insufficiency, arrhythmia, etc. Hemorrhages, especially epistaxis, but also hemoptysis, hematemesis, etc., sometimes occur. Muscular tremors, fine, rhythmic, 8 to 10 to the second, are present in almost all cases and are noteworthy in more than one-half of the cases. There is often also marked muscular weakness; sometimes a peculiar giving way of the legs (Charcot).

There are many vasomotor symptoms, such as subjective complaint of warmth, and itching, dermographism, erythema, urticaria, angioneurotic œdema, readily provoked and excessive flushing and sweating, diarrhœa, sometimes vomiting and abdominal pain, periodical polyuria, cough, dyspnœa, intermittent (sometimes painful) swelling of the joints, etc. Often there are nervous symptoms, such as headache, vertigo, insomnia, different forms of paralysis, mental irritability or depression; occasionally, especially in the acute cases, psychoses. The skin reveals lessened resistance to the galvanic current (Chvostek); it may become pigmented or reveal spots of vitiligo; the hair may fall. Attacks of fever, remittent or intermittent, lasting several days or several weeks, are not uncommon. The metabolism is increased (excessive nitrogenous and other excretion).

Eventually emaciation, cardiac weakness (myocardial degeneration), low blood pressure, and more or less secondary anemia supervene. There may be a relative lymphocytosis (usually no leukocytosis; sometimes leukopenia), and the coagulability of the blood may be lessened. Unusual and rapid loss of weight often attends acute exacerbations of the disease (fever, vomiting, diarrhœa, marked vasomotor disturbances, extreme tachycardia, arrhythmia, perhaps mania, etc.); the loss may be partly compensated for by gain during a succeeding period of remission. Death may ensue from acute thyroidism, cardiac weakness, exhaustion from vomiting and diarrhœa, secondary infection (tuberculosis, pneumonia, etc.), diabetes (which sometimes ensues), etc.

Diagnosis.—The fully developed disorder presents no diagnostic difficulties, but incomplete cases, slight or moderate hyperthyroidism, often go unrecognized. Attention to other than the three (formerly)

so-called cardinal symptoms (goitre, exophthalmos, and tachycardia) will often enable one to make the diagnosis in early stages and in aberrant cases. Tachycardia and the vasomotor phenomena (which determine the severity of the disease) with or without slight turgescence of the thyroid, are of great diagnostic significance. One must not mistake the slight and temporary enlargement of the thyroid that may follow shocks and emotional disturbances, or occur at puberty, during pregnancy, and the climacteric, for Graves' disease, although these may progress to the most serious disorder, and time alone may be required for demonstrative differentiation. Nor should the enlargement of anemic states, especially chlorosis, and of some forms of heart, especially mitral, disease be mistaken. The hyperthyroidism of early tuberculosis (sometimes associated with tuberculosis of the thyroid), neurasthenic states in which transitory thyroid enlargement is not uncommon, etc., must also be excluded. In doubtful cases recourse may be had to the use of iodine internally or some thyroid tablet: in the event of hyperthyroidism even small doses readily provoke tachycardia and other characteristic symptoms (intolerance of the hyperthyroid subject).

Prognosis.—The disease usually runs a protracted course, extending over many years—twenty or more. Spontaneous recovery in well-developed cases is rare; but mild cases or moderate grades of hyperthyroidism may be much benefited, if not completely cured, by appropriate treatment. Marked benefit, if not cure, may be expected in at least half of the cases. Cases that develop acutely, or acute exacerbations of chronic cases, may lead to death within a few weeks to a few months.

Treatment.—Rest with freedom from care, worry, and excitement are of prime importance; in severe cases, rest in bed for a month or two is essential; and in all cases a change of scene and air is beneficial. Careful attention must be directed to the diet, so that the patient may be well nourished. Thomson (who believes the disease to be a toxemia originating in the intestinal tract) urges the use of fermented milk (keffir) or peptonized milk in large amounts, vegetables (except peas, beans, and asparagus), crusty bread, rice, and fruits (except uncooked apples and strawberries), and the prohibition of butcher meat, oysters, clams, and lobsters; one egg daily may be permitted, and a small amount of non-oily fish, poultry, quail, or partridge may also be taken. The bowels must be opened daily; paroxysms of diarrhœa may require the use of astringents, which, however, are rarely very useful. General hydrotherapy, as well as cold applications to the thyroid and the heart are very useful. The cardiovascular and vasomotor phenomena are perhaps best controlled with belladonna (or atropine) given to the point of toleration; the bromides and other nervines, or small doses of opium for short periods, are sometimes useful adjuncts, but opium should be given with the greatest circumspection, or not at all, to subjects in whom the prognosis is relatively good. The hyperactivity of the heart is sometimes benefited by aconite

or veratrum viride; little benefit attends the use of digitalis, strophanthus, convallaria, etc., unless there is actual disease of the heart; otherwise arrhythmia, etc., may be made worse. A number of drugs have been more or less highly extolled from time to time. Arsenic is believed to exercise a modifying influence on the secretion of the thyroid: to secure results it must be given for a long time; phosphorus, especially in the phosphates, also is credited with some action on the thyroid secretion, but it is doubtful if, as usually given (sodium phosphate), it exercises any virtues other than those attending free evacuation of the bowels; quinine hydrobromide (the neutral salt) to the point of toleration for a long time is said to exert beneficial, even curative, properties. In addition to sodium phosphate before each meal and a blue mass pill twice weekly, Thomson uses intestinal antiseptics, such as sodium salicylate and sodium benzoate, of each 10 grains (0.6 gram) one hour after each meal, and a capsule of naphthelene 3 grains (0.2 gram) and sodium benzoate 6 grains (0.4 gram) at bedtime; phenol bismuth and ammonium benzoate also are used. Iron is of value in anemic subjects. Ergot, adrenalin, electricity, the *x*-rays, etc., have been advocated from time to time; but they are of doubtful value. Iodine, the iodides, and thyroid extract are, as a rule, harmful. The serum of thyroidectomized dogs, sheep, and goats, and the milk of thyroidectomized goats, and extract of the thymus, have been used in the belief that they contain specific antibodies; but great success seems not to have attended their use. The cytolytic serum of Rogers and Beebe seems to be definitely curative in some cases, but further experience in its use is desirable. Surgical treatment (resection of part of the thyroid; ligation of the superior thyroid arteries) is indicated in patients who do not improve under well-directed medical treatment, including rest, in from three to six months, in those who become progressively worse under medical treatment, and in those with signs of local pressure.

HYPOTHYROIDISM; CRETINISM; MYXŒDEMA.

Deficiency of the thyroid secretion (hypothyroidism, athyrea), may give rise to one of three conditions: (1) Cretinism; congenital or acquired myxœdema of infants and children; (2) spontaneous myxœdema of adults; and (3) postoperative myxœdema.

Cretinism.—Cretinism is a disorder due to lack of thyroid secretion and characterized by imperfect development of the body and the mind. It occurs as an endemic and a sporadic affection. Endemic cretinism is common in endemic goitrous regions. One or both parents may be goitrous, or mildly cretinoid; although if both parents are cretins the marriage is usually sterile. The thyroid may be absent or small, sometimes normal in size or enlarged; the epithelium is atrophic and the colloid is increased. Sporadic cretinism occurs in non-goitrous regions, and may result from aplasia or hypoplasia of the thyroid, thyroiditis (infec-

tions with consecutive sclerosis or destruction of the thyroid parenchyma). The lesions are quite the same as those of endemic goitre. The thymus often persists.

Symptoms.—Usually toward the end of the first or during the second year arrest of bodily and mental development becomes apparent (infantile form); occasionally the symptoms are delayed for several years (juvenile form), although they always appear before puberty; but the earlier the onset the more marked the phenomena. The head is large, the fontanelles are open, the forehead is low and broad, the nose is flat, the mouth is usually open, the lips are thick and through them protrudes a thickened tongue; the expression is stolid, idiotic, or quite bestial. The body is stunted, dwarfed, the limbs and the neck are short and thick, the skeletal muscles small and inefficient, and the gait, if the child walks at all, weak and waddling; and the abdomen is large and protuberant. The skin is pallid, dry (no sweating), thick, inelastic, and seemingly œdematous (but does not pit on pressure—myxœdematous); the hair is thin and scanty, the nails brittle, and the teeth carious. The body temperature is low, the pulse infrequent, and metabolism deficient. The child is more or less idiotic, difficult or impossible to train; speech is often limited to a few more or less inarticulate words; deafness may be present; the genitals are retarded or do not develop at all. Cretins may live forty, fifty, or sixty years.

Diagnosis.—A cretin is usually recognizable at sight; but in some cases the phenomena are not marked (semicretins; moderate hypothyroidism) and the condition may be overlooked. Achondroplasia (fœtal rickets, fœtal chondrodystrophy), a peculiar form of dwarfism, may be distinguished by marked shortening of the extremities (micromelia), great enlargement of the joints (hyperplasia of the cartilages), and absence of myxœdema, cretinoid expression, and defective mentality. Infantilism is characterized by an infantile condition of the genitalia and the absence of secondary sexual characteristics, but there is no myxœdema or other cretinoid characteristics. Idiocy, hydrocephalus, etc., should be readily distinguished.

Prognosis.—The disease is progressive without specific treatment. Death often occurs early in life, but if the subject survives a few years, life may be prolonged forty or more years.

Myxœdema of Adults.—Myxœdema of adults is a disorder occurring spontaneously in adults, due to a lack of thyroid secretion, and characterized by myxœdema, physical weakness, and mental deterioration. The disorder is six to seven times as common in women as in men, and is most common in adult life (thirtieth to the fiftieth year). It may be hereditary or familial; or one member may have exophthalmic goitre and another myxœdema, etc. The thyroid is usually atrophic or absent; if of normal size or enlarged, the epithelium is atrophic and degenerated. The thymus and the pituitary have been found enlarged.

Symptoms.—The onset is usually insidious. The patient is believed to be "taking on flesh;" but the skin becomes harsh, dry, pale, inelastic,

thickened, and apparently œdematous (but does not pit on pressure —myxœdema). Thick pads of such tissue may develop on the abdomen, buttocks, thighs, supraclavicular regions, etc. The face becomes broad, swollen, and coarse, the lips, nose, and tongue thick, and the expression dull and listless; there is often a characteristic flush on the cheeks. The gait has been likened to that of a hippopotamus. Physical weakness progresses and may become extreme; and to this is joined mental apathy, slowness of thought and speech, defective memory, somnolence, and eventually dementia (which may be preceded by delirium, mania, etc.). The temperature is usually subnormal. There may be various paresthetic phenomena or actual neuromuscular pains (especially in the calves); undue sensitiveness to cold; arteriosclerosis with hypertrophy and dilatation of the heart; indigestion and constipation; albuminuria or glycosuria; uterine and other hemorrhages; anemia, etc.

Postoperative Myxœdema.—Cachexia strumipriva is a myxœdematous condition with the attendant physical and mental deterioration that develops after complete and sometimes after partial removal of the thyroid body. The amount of gland necessary to prevent this form of myxœdema has not been determined, but a small part, less than one lobe, usually suffices. The symptoms are quite the same as those of spontaneous myxœdema in the adult.

Diagnosis.—Myxœdema should be recognized readily by attention to all the phenomena, cutaneous, physical, and mental. Obesity should be readily excluded. Bright's disease, suggested by the pallor and subcutaneous swelling, perhaps albumin and casts, should be readily excluded by the absence of pitting on pressure, and by the thickness, induration, and dryness of the skin, loss of hair, subnormal temperature, marked asthenia, and the mental phenomena. Incomplete or mild cases are likely to be overlooked, being mistaken for neurasthenia, psychasthenia, senile dementia, etc.; but careful investigation will disclose the asthenia, the mental apathy, the myxœdematous condition of the skin, the small thyroid, etc.

Prognosis.—The disease is steadily progressive in the absence of specific therapy.

Treatment.—Deficiency of thyroid secretion, cretinism, and myxœdema are imperative indications for the use of thyroid preparations—a specific treatment. Usually the thyroid of sheep is used, beginning with one grain (0.06 gram) thrice daily and gradually increasing to fifteen grains (1 gram) or more a day or until the point of toleration (thyroidism) is reached (nervousness, cutaneous flushing, paresthesias, muscular twitchings or tremors, tachycardia, and other phenomena suggesting exophthalmic goitre). The effects are inconceivable to those unacquainted with them. After cure or marked improvement has been effected by large doses, smaller doses must be continued to insure permanency of results and prevent relapse. Good food, good hygiene, fresh air, hydrotherapy, and tonics (iron, arsenic, etc.) are useful adjuvants.

TUMORS AND CYSTS OF THE THYROID.

Tumors of the thyroid may be benign (?) or malignant—adenoma, carcinoma, and sarcoma. An adenoma develops as a solid, encapsulated, usually soft, and smooth, growth in one lobe of the thyroid, compressing the normal thyroid tissue. It may be of the fœtal type—solid, close vesicles, with little connective tissue stroma, and no colloid; or of the mixed type—solid vesicles without, and large, distended vesicles, with colloid; or of the papilliferous cystic type—cystic vesicles with papilliferous ingrowths. In addition to cystic formation (softening) these growths are prone to hemorrhage; and that they are not benign, or at least do not remain benign, is evident in the fact that they (especially the papilliferous cystadenoma) may give metastases; and that they proceed by gradual histological variations to definite carcinoma. Sarcoma is rare, and may be of any of the common types. As a rule, tumors remain for a considerable time unsuspected, being not distinguished (and often indistinguishable) from simple goitre (hypertrophy); but malignant goitre should be diagnosticated in unilateral enlargements in adults, especially if the growth is rapid and nodular. The diagnosis, however, often is not made until the growth has penetrated the capsule, formed adhesions to adjacent tissue, and given rise to metastases to the regional lymph nodes or to distant parts, such as the lungs and the bones (a solitary bony metastasis—inferior maxilla, skull, sternum, etc.—being quite characteristic). In the differential diagnosis one must exclude the rare large solitary tubercle and a gumma.

In addition to the softening and hemorrhagic cysts already mentioned, the thyroid may be the seat of echinococcus cyst. Aberrant or accessory thyroids are sometimes encountered at the base of the tongue, in the mediastinum, pleura, etc., and by proliferation may give rise to tumor formations.

DISEASES OF THE PARATHYROID GLANDS.

Pathological Physiology.—The parathyroid glands (or bodies) consist, as a rule, of two pairs, upper and lower (four glands, sometimes six or eight), somewhat flattened and ovoid in shape, from 6 to 8 mm. long by 1 to 3 mm. in diameter, situated along the posterior inner edge of the lateral lobes of the thyroid. The physiological activities of the parathyroids have not yet been completely worked out; but the glands seem to be essential to life and to be intimately concerned in the calcium metabolism. Operative removal, as has been demonstrated in the lower animals, and in human subjects (unintentional removal or deprivation of blood supply when removing the thyroid), results in tetany—hypoparathyreosis or cachexia parastrumipriva (Halsted). The phenomena may be ameliorated by feeding parathyroid preparations, and by injections of calcium

salts; and they may be prevented by autotransplantation of the parathyroids (Halsted). The parathyroids are doubtless in some unknown way related to the other ductless glands (through the medium of hormones). They seem also to exercise some influence (antagonistic to that of the thyroid) on carbohydrate metabolism: following extirpation of the parathyroids, adrenalin glycosuria is readily produced, that is, the limit of carbohydrate metabolism is reduced.

The parathyroids have been supposed also to be concerned in other convulsive disorders, such as epilepsy, myoclonia, myotonia, paralysis agitans, but upon insufficient evidence. Degeneration and sclerosis of the parathyroids have been found in infantile atrophy (R. L. Thompson). Tumors occasionally occur. Parathyroid preparations have been used with success in parathyroid insufficiency (tetany); and without success in Graves' disease, myxœdema, convulsive disorders, etc.

TETANY.

Tetany is a disorder characterized by bilateral tonic, intermittent or continuous spasms of the extremities, and increased mechanical and electrical excitability of the peripheral nerves.

Etiology.—Tetany sometimes occurs endemically or epidemically in certain parts of Europe (Berlin, Vienna, etc.), but it is rare in this country. In Europe it is especially common in young men of the artisan class, such as shoemakers, tailors, etc. It sometimes occurs in infections, such as typhoid fever, scarlatina, measles, cholera, etc.; in intoxications, such as chloroform, lead, and ergotin poisoning, uremia, etc.; in gastro-intestinal disorders, such as dilatation and other disorders of the stomach with retention; and in pregnancy and during lactation (nurse's contracture, Trousseau). It also follows operation upon and excision of the thyroid (removal or destruction of the parathyroids). In children it is usually associated with rickets, but sometimes also with acute infections, gastro-intestinal disorders, etc.

The nature of the disorder is still somewhat imperfectly understood, but the researches of McCallum and Voegtlin and others suggest that it is due to insufficiency of the parathyroids and disturbance of the calcium metabolism. The parathyroids in some ill-understood way control the calcium exchanges in the body, and the absence or perversion of the parathyroid secretion leads to impoverishment of the tissues as regards calcium, perhaps by permitting calcium to combine with other substances and thus to be abstracted from the tissues. The result of this calcium impoverishment is hyperexcitability of the nerve cells and tetany; so that tetany may be regarded as a state of hyperexcitability of the nerve cells due to calcium impoverishment.

Symptoms.—The chief symptom consists of bilateral, tonic, painful, intermittent, or continuous spasms of the muscles of the hands (accoucher's hand), and less frequently of the feet (equinovarus position);

rarely the wrists, arms, face, legs, etc., may be involved. There is also increased mechanical excitability of the nerves: tapping the facial, median, ulnar, perineal nerves, etc., serves to provoke contracture (Chvostek's sign); and compression of the nerves provokes the same result (Trousseau's sign). The attacks continue for a variable time: in children often only an hour or two, in adults sometimes for a week or two—with exacerbations and remissions. There is also increased electrical excitability of the nerves: of the motor nerves to the galvanic current (Erb's sign), of the sensory nerves (Hoffmann's sign), and of the nerves of special sense (F. Chvostek's sign). The sensorium, as a rule, is not involved, and there are no objective sensory changes. In severe, acute cases there may be fever. Laryngismus stridulus sometimes occurs also in rickety children.

Diagnosis.—The peculiar bilateral spasms and the increased mechanical and electrical excitability of the nerves are quite pathognomonic.

Prognosis.—The prognosis varies with the associated conditions, being serious in operative tetany, in gastric tetany, and in laryngismus stridulus.

Treatment.—The treatment varies with the etiological factors. In rickets, infections, and intoxications, the treatment is that of the underlying cause. Weaning the child often cures the cases occurring during lactation. In the gastric cases lavage with saline and mild antiseptic solutions is sometimes efficacious. In operative tetany benefit accrues from the use of parathyroid preparations, and perhaps also from the subcutaneous transplantation of parathyroid bodies. The acute symptoms are effectively controlled by the intravenous injection (less effectively by the subcutaneous injection or ingestion) of 5 per cent. solution of calcium lactate (or acetate), in physiological saline solution—1000 to 2000 c.c. at a time; magnesium salts also may be used. Chloral, bromides, and morphine may serve as useful adjuvants.

DISEASES OF THE ADRENALS AND THE CHROMAFFIN SYSTEM.

Pathological Physiology.—The adrenals, composed of readily differentiated cortex and medulla, are partly of epithelial and partly of nervous origin. The cortex in all probability is derived from the cœlum epithelium; the medulla is derived from the sympathetic nervous system, and consists in large part of peculiar polymorphous cells, which in chromic acid solutions (chromic acid salts) take on a brownish pigmentation, and are, therefore, spoken of as chromaffin cells. Similar cells are found elsewhere in the sympathetic nervous system and its derivatives—the ganglia and larger nerve trunks of the sympathetic, as well as in certain so-called paraganglia, especially along the course of the aorta near its bifurcation (Zuckerkandl's bodies), the carotid glands, the coccygeal gland, collection of cells in the parovarium, in the epididymis, etc.—

all of which collectively are spoken of as the chromaffin system. Similar cells have been found also in aberrant adrenal rests and in the pituitary body.

The functions of the adrenals are not completely known. The organ seems to be essential to life; removal in lower animals soon leads to death in the picture of severe intoxication. The cortex is believed to exercise a detoxifying function, neutralizing circulating poisons of intermediate metabolism; and to control, at least in part, growth and development, especially of the sexual organs (the adrenals of animals enlarge during sexual activity and pregnancy; cortical hyperplasia and tumor formations are sometimes associated with precocious sexual development, and hypoplasia with retarded sexual development). The medulla and the chromaffin system throughout the body contain adrenalin, which may be looked upon as the specific product of the chromaffin system. Its chief function is the maintenance of blood pressure (through its stimulating action upon the vasoconstrictor fibers of the sympathetic) and of muscular tone, perhaps by furthering oxidation in the tissues. It probably exercises some influence on carbohydrate metabolism, since subcutaneous or intravenous injections give rise to glycosuria. The chief evidence of disordered function is cardiovascular and muscular asthenia (adrenal insufficiency), which is most marked in Addison's disease. Less grades of such asthenia are common in many exhausting and debilitating diseases, and have been attributed to adrenal insufficiency. Some of these, such as pulmonary tuberculosis, gastric carcinoma, etc., may be associated with increased cutaneous pigmentation (as in Addison's disease), and others, such as neurasthenia, etc., with low blood pressure. In some infections (diphtheria) and other disorders, the myasthenia may come on suddenly and be very severe, and may be associated with lessened blood pressure; the condition may be relieved by adrenalin; in some fatal cases the adrenals have been found lacking in adrenalin, so that a condition of acute adrenal insufficiency has been postulated. Less severe cases are spoken of as subacute and chronic adrenal insufficiency (incomplete Addison's disease). Whether an excess of adrenalin is ever formed is not known, but the well-known fact that adrenalin provokes arteriosclerosis in the lower animals has led to the supposition that it may be active in the causation of arteriosclerosis in man, and also to the suggestion that the metabolic products retained in the body in renal disease stimulate the adrenal medulla to increased functional activity, and that the increased adrenalin thus formed is the cause of the excessive blood pressure. Perhaps through some other influence an excess of adrenalin may be formed (or adrenalin may not be excreted), and lead not only to the heightened blood pressure, but also to the renal and cardiac changes of chronic cardiovascular-renal disease.

Preparations of the adrenal (adrenalin) injected intravenously or subcutaneously are valuable in increasing the blood pressure in shock and conditions of low vascular tension generally (such as infections, intoxi-

cations, etc.); and they are of service in controlling hemorrhages from surfaces to which they can be applied locally.

Aside from Addison's disease and tumor formation, the adrenals are rarely the seat of disease of clinical interest. Degenerative changes (cloudy swelling, fatty and amyloid degeneration) accompany similar conditions in other organs. Hemorrhages occur in the newborn (especially after difficult labors and still births), traumatisms, toxic and infective processes. They may give rise to pain in the upper abdomen, vomiting, collapse, convulsions, tympanites, diarrhœa, etc., and death. Tumors (hypernephromas) partake of the characteristics of retroperitoneal growths. Tumors of adrenal tissue also are common in the kidney (hypernephroma being the most common tumor of the kidney in the adult), and elsewhere along the genito-urinary (especially the female genital) tract.

ADDISON'S DISEASE.

Addison's disease is a disorder due to adrenal inadequacy (hypoepinephry) characterized anatomically, as a rule, by tuberculosis of the adrenals and degenerative lesions in the chromaffin system elsewhere, and clinically by muscular and vascular asthenia, gastro-intestinal irritability, and pigmentation of the skin and mucous membranes.

Etiology.—The disorder is most common in young adults (before the fortieth year), and is almost twice as common in males as in females. The etiological factors are those of tuberculosis and of tumor formation in general, and are as ill understood.

Pathology.—Usually there is fibrocaseous tuberculosis of the adrenals —unilateral or bilateral; less commonly there is simple atrophy with associated fibrosis, or tumor formation. In addition, as Wiesel has pointed out, the chromaffin cells of the sympathetic nervous system (adrenal medulla, abdominal plexuses, etc.) disappear; the cortical substance of the adrenals may be preserved, as well as accessory adrenals, if any be found. Many of the ganglion cells exhibit not only the lipochrome pigmentation (which is not specific), but also the specific chromaffin reaction (characteristic of Addison's disease). Wiesel believes Addison's disease to be a specific affection of the chromaffin system, consisting of successive atrophy of the chromaffin tissue, the process involving the adrenal medulla and spreading to the cortex; to compensate for this loss of chromaffin tissue some of the ganglion cells become chromaffin. The cases of extensive adrenal tuberculosis without clinical signs of Addison's disease are explained on the basis of absence of general disturbance of the chromaffin system; and the cases of Addison's disease without disease of the adrenal are believed to be due probably to the fact that lesions of the chromaffin system outside the adrenal are sufficient to produce the symptom-complex of the disease.

Symptoms.—The following is adapted from Addison's classical description: The patient is observed gradually to fall off in general

health; he becomes languid and weak, indisposed to bodily or mental exertion; the appetite becomes impaired or entirely lost; the whites of the eyes become pearly, the pulse small and feeble, or perhaps somewhat large, but excessively soft and comprehensible; the body wastes, without, however, presenting the dry and shrivelled skin and extreme emaciation usually attendant on protracted malignant disease; slight pain or uneasiness is, from time to time, referred to the region of the stomach, and there is occasionally actual vomiting which may be both urgent and distressing; and the patient may manifest indications of disturbed cerebral circulation. Gradually a characteristic discoloration of the skin becomes apparent; this pervades the whole surface of the body, but is commonly most marked on the face, neck, arms, penis, and scrotum, and in the flexures of the axillæ and around the navel; it presents a dingy or smoky appearance or various tints of deep amber or chestnut-brown; it may occur in patches, or certain parts may be so much darker than others as to impart to the surface a mottled appearance; and in the midst of this dark mottling certain insular portions of the skin may present a blanched or morbidly white appearance (actual defect of pigment), contrasting strongly with the surrounding skin. Similar irregular distribution of pigmentation may occur in the mucous and serous membranes. This singular discoloration usually increases with the advance of the disease; the anemia, languor, failure of appetite, and feebleness of the heart become aggravated; the body wastes; the pulse becomes smaller and weaker; and without any special complaint of pain or uneasiness the patient gradually sinks and expires.

Diagnosis.—Muscular, vascular, and mental asthenia, pigmentation of the skin and visible mucous membranes, and gastro-intestinal symptoms (pain, vomiting, and diarrhœa) are quite diagnostic, especially when, as stated by Addison, "neither the most diligent inquiry nor the most careful physical examination tend to throw the slightest gleam of light upon the precise nature of the patient's malady." The maximum blood pressure may be 100 mm. Hg. or less. The blood shows the ordinary changes of secondary anemia (which excludes pernicious anemia). Other sorts of pigmentation must be excluded, such as that of dark-skinned races; pregnancy and disease of the female genitalia; vagabond's disease (pigmentation of exposed parts; rough and scratch-marked skin; often associated pediculi); pernicious anemia; Graves' disease; diseases of the stomach, especially carcinoma; chronic jaundice; hemochromatosis (bronzed diabetes); chronic malaria; pellagra; melanosarcoma of the skin; chronic poisoning with silver nitrate (argyria), arsenic, etc. It is wise to withhold the diagnosis in the absence of asthenia. In case of doubt resort may be had to tuberculin tests, but positive results must be interpreted cautiously, since they may be due to tuberculosis elsewhere than in the adrenals.

Prognosis.—The disease is doubtless invariably fatal. The common duration is about three or four years from the onset of symptoms, but this may be influenced by tuberculosis elsewhere in the body. Some-

times remissions or standstill ensue and the patient may live ten or more years. Rarely there are what are called acute cases (acute adrenal inadequacy), in which, with marked asthenia, vomiting, diarrhœa, and the general evidences of severe intoxication, death may ensue within a few weeks.

Treatment.—The treatment is almost wholly symptomatic: rest in bed on account of the asthenia and a tendency to marked and perhaps fatal syncope; good nutritious diet—especially milk; strychnine and arsenic as general tonics; bismuth, creosote, carbolic acid, cerium oxalate, dilute hydrocyanic acid, etc., for the gastro-intestinal irritability. The use of adrenal preparations by the mouth, subcutaneously, and intravenously, is indicated theoretically; practically their use has been attended by diverse results: they seem to be of much value early, in doses to suit the exigencies of individual cases. If a tumor or salivary tubercle of the adrenal can be diagnosticated it should be removed.

DISEASES OF THE PITUITARY BODY.

Pathological Physiology.—The pituitary body, or hypophysis cerebri, is composed of two lobes—an anterior, the larger, of epithelial origin (a diverticulum of the buccal epithelium), and a posterior, smaller, in part of nervous origin (infundibular pouch of the thalamencephalon) and in part of epithelial origin, the two being separated by a persisting cleft. The physiological properties of the pituitary body have not been definitely determined, although the body seems to be essential to the maintenance of life. The posterior lobe (doubtless the nervous nucleus) contains a blood-pressure-raising substance (pituitarin) and a diuresis-producing substance (increasing the blood pressure or acting directly on the renal epithelium). The anterior (perhaps more important) lobe is believed to exercise some influence over bodily growth and sexual development. Augmented function (hyperpituitarism), such as may occur in hypertrophy and tumor formations, and is perhaps referable to increase in number or overactivity of the chromophile cells (less likely subactivity of the chromophobe cells), seems to lead to gigantism if it occurs during early life, and to akromegaly if it occurs during adult life. Deficient function (hypopituitarism) occurring in early life seems to lead to certain types of infantilism associated with excessive deposition of fat and imperfect development of the sexual organs and of the secondary sexual characteristics; perhaps the loss or retrogression of these sexual characteristics in adult life may be due also to hypo-activity of the pituitary. Removal of the gland in lower animals has led to the development of a condition comparable to that believed to be due to hypopituitarism in man; and excision of the enlarged body in akromegaly had been followed by notable improvement (Cushing). The pituitary also bears some relationship to the other ductless glands: it has been found enlarged after excision or disease of the thyroid and

adrenal, and it (especially the posterior lobe) is said to contain a substance (hormone) that stimulates the adrenal and inhibits thyroid activity, while the anterior lobe is said to stimulate thyroid activity.

AKROMEGALY.

Akromegaly is a disease, probably due to disordered function of the pituitary body, and characterized by abnormal growth especially of the face, hands, and feet.

Etiology.—Akromegaly is about equally common in the two sexes, and is most common in the third decade (75 per cent. of the cases); it is rare before the twentieth and after the fortieth year. There is no known cause, ascribed factors, such as shock, fright, alcoholism, syphilis, etc., being of more than doubtful significance.

Pathology.—The majority of cases exhibit some more or less obvious disease of the pituitary, notably tumor formation (adenoma, carcinoma, sarcoma), but sometimes softening, hemorrhage, and fibrosis; microscopic lesions have been found in some pituitary bodies normal to the unaided eye. The bones of the skull, and those of the hands and feet, especially, but also the other bones of the body to a greater or less extent, become enlarged, thickened, and denser than normally. The enlargement is due to periosteal overgrowth, and involves particularly the bony prominences. The soft tissues of the extremities also become thickened—by connective tissue hyperplasia and fibrosis (answerable for much of the enlargement). The other ductless gland sometimes shows divers, but no constant, changes. The viscera (heart, liver, spleen, kidneys, etc.) are sometimes enlarged. The pancreas may be fibrosed. The internal genitalia may be enlarged, but the testicles, uterus, and ovaries, usually are atrophic or degenerated.

Symptoms.—The bony enlargements are sometimes the earliest symptoms, but in the majority of cases antecedent symptoms referable to the overgrowth of the pituitary (brain tumor) may be elicited: headache, vertigo, vomiting, mental irritability and other alterations, disturbance of the special senses (sight, hearing, and taste), and of the cranial nerves (optic neuritis, oculomotor and other paralyses). These, if not developed early, are likely to supervene later. In addition there may be paresthesias, more or less severe pains, etc., in the extremities. The characteristic of the disease is abnormal growth, especially of the face, hands, and feet—which, although much enlarged, are not, as a rule, otherwise deformed. The head increases in size, but less so than the face, which in consequence of marked growth of the superior and (especially) the inferior maxilla, becomes elongated. The teeth become separated, from widening of the alveolar arches. The hands and feet show one of two types—the long or the thick (spade-like, sausage-like fingers). The hypertrophy involves also the soft parts of the hands and feet, and face (nostrils, lips, eyelids, cheeks, ear, tongue, etc.). Later, other bones (ster-

num, ribs, vertebræ, etc.) may become involved, and kyphosis, etc., may supervene. The skin, in general, may become harsh, dry, and thickened (perhaps remotely suggesting myxœdema), or there may be profuse sweats; sometimes also the mucous membrane of the mouth, nose, larynx, etc., becomes thickened. The muscles may become atrophic, the huge frame and bulk of the subject then contrasting markedly with the general muscular weakness. Sexual disorders are common—amenorrhœa in women, impotence in men. Glycosuria sometimes supervenes. Arteriosclerosis and hypertrophy and dilatation of the heart are common. The viscera (liver, spleen, and kidneys) may become enlarged, and the lymph nodes and the thymus may become palpable.

Diagnosis.—The disease is usually recognized on sight. Especial attention should be paid to bitemporal hemianopsia, optic neuritis or atrophy, oculomotor paralyses, and other symptoms of tumor in the sella turcica, as early signs. Gigantism, osteitis deformans, and hypertrophic pulmonary osteo-arthropathy must be differentiated.

Prognosis.—The disorder is progressive, but often lasts for many (twenty or more) years. Death results from general asthenia, secondary infection, brain tumor, cardiac weakness, diabetes, etc.

Treatment.—There is no known efficacious medical treatment. Operative removal of at least a part of the enlarged pituitary appears to be the treatment of the future; it has already been successfully done with benefit by Cushing.

SECTION V.

DISEASES OF THE BLOOD AND HEMOPOIETIC SYSTEM.

Pathological Physiology.—The blood is a tissue as well as a fluid—a tissue reflecting the state and condition of the so-called hemopoietic organs, of which it is the circulating portion; and a fluid containing the soluble products of the metabolic activities of all the component parts of the body. Morphologically it is of comparatively simple constitution, consisting of erythrocytes, leukocytes, platelets, and plasma; but chemically it is of exceedingly complex composition. It subserves primarily the function of conveying to the body-cells oxygen and absorbed food products, essential to their life and well being, and of receiving and disposing of the products of cellular activity, whether these be refuse or of further use in the economy; but, in addition, the blood is charged with another most important function—that of defense against divers infectious and toxic processes to which the body is constantly exposed. Thus, it represents the sum total of the activity and inactivity, the anabolism and katabolism, and normal and perverted function of all the tissues and organs of the body, of which it is the connecting link.

So subject is the composition of the blood to the influences of extra-circulatory conditions that disorders of the blood must be largely, if not wholly, merely a reflection of diseased processes elsewhere. Is the body anywhere diseased, so also is the blood; divers toxic processes, such as accompany infections, intoxications, and disorders of metabolism, like diabetes, gout, uremia, etc., are in a sense disorders of the blood: so that one may well doubt whether the blood is ever primarily diseased. In a general sense also one may say that the hemopoietic system comprises all the tissues and organs of the body, since each contributes its moiety to the composition of the whole. But since custom and convenience sanction a separate discussion of disorders in which changes in the blood are noteworthy, so also they sanction restriction of the term hemopoietic organs to those concerned in the production of the formed elements of the blood.

In the fœtus this hemopoietic system consists of the bone marrow, the lymph nodes, the spleen, and the liver, and there is no sharp line of

differentiation between the tissues producing erythrocytes, on the one hand, and leukocytes, on the other. At birth the liver has lost its blood-forming function; and as growth continues the other hemopoietic organs gradually become specialized, so that in adult life the erythroblastic and the leukoblastic tissues are sharply differentiated, although under the stress of circumstance they may revert to the fœtal type. In adult life the erythrocytes are formed in the red marrow of the bones by division or loss of the nuclei of the erythroblasts, which in turn, perhaps, are derived from colorless, mononuclear, non-granular mother cells that take on hemoglobin; it is believed that in some diseased states the hemolymph nodes also may form erythrocytes. The granular leukocytes are formed in the bone marrow, being derived from certain mononuclear neutrophilic, eosinophilic, or basophilic cells, which in the course of time, and usually before they enter the circulation, develop polymorphous nuclei, and give rise to the polynuclear neutrophiles, eosinophiles, and basophiles (so-called mast-cells) of the circulating blood. The origin of the so-called mononuclear leukocytes is still in dispute, although the weight of evidence assigns them to the bone marrow; perhaps they are mother cells. The lymphocytes are formed in the lymph nodes, thymus, tonsils, and lymphoid tissues generally throughout the body. In the central portions of the so-called germ centres, where proliferation is active, the cells are large and correspond with the large lymphocytes of the circulating blood, but toward the periphery of the germ centres, the cells are smaller and correspond with the ordinary small lymphocytes of the circulating blood. Whether any circulating lymphocytes are formed in the bone marrow in health is not definitely known, although it seems likely; but in diseased states, such as leukemia, the marrow contributes large numbers of so-called lymphocytes—probably not identical with the circulating lymphocytes of health. There is considerable evidence that both the red and the white cells are derived from a common ancestor—a large mononuclear, non-granular, somewhat basophilic marrow cell; but when once started on their life journey, cells of the one series cannot become converted into the other. The blood platelets, according to the studies of J. H. Wright, are formed by detachment of the pseudopod-like processes of the giant cells (megakaryocytes) of the bone marrow.

The erythrocytes are designed to absorb and diffuse oxygen and perhaps carbon dioxide—their sole known function—which they effect by reason of their contained hemoglobin, and for which their flexibility and biconcave surface admirably adapts them. The functions of the leukocytes are ill understood: endowed with independent motility, they appear to be concerned in the absorption of fat and protein from the intestine and in other nutritive (cellular) processes; they contribute to the clotting of blood by producing fibrin ferment, thrombokinase; they produce also apparently a fat-splitting and a tryptic ferment; they facilitate the repair of injuries; they are especially concerned in various defensive processes (phagocytosis) whereby the body is enabled to resist bacterial invasion; and they act as scavengers in removing animate and

inanimate foreign substances that act as sources of irritation. But their function is doubtless more comprehensive. The polynuclear leukocytes are those especially concerned in phagocytosis; the eosinophiles, while non-phagocytic are especially concerned in parasitic infections; the mast-cells and the lymphocytes also doubtless have specific functions, although they are unknown. The blood platelets are much concerned in coagulation of the blood (whence they have been termed thrombocytes); they are diminished in conditions, such as pernicious anemia, purpura, typhoid fever, etc., in which clotting is poor and the bone marrow deficient in megakaryocytes.

The blood doubtless varies considerably in composition from time to time under various influences, but the remarkable fact is its tendency to maintain a uniform composition under many and varied conditions. Normally the erythrocytes number about 5,000,000 to the cubic millimeter in man, and 4,500,000 in women; the leukocytes, 6000 to 10,000 (averaging about 7500); and the platelets, 200,000 to 700,000. The plasma also tends to maintain a uniform composition by the excretion of deleterious substances through the kidney, skin, and lungs, and, as occasion demands, absorbing from or discharging into the lymph channels some of its substance.

The circulating blood corpuscles are believed to enjoy only a brief life career. The erythrocytes are credited with an average life of three or four weeks, but this is not susceptible of proof. They probably gradually succumb to the stress of a strenuous existence, and are finally completely destroyed in the spleen and liver, perhaps even in the vascular channels, and at least in certain diseased states in the hemolymph nodes. The life span of the leukocytes is not known; they are believed to succumb even earlier in circulatory life than the erythrocytes, and to find their *campo santo* in the spleen.

Anemia.—What otherwise would be an early dearth of blood corpuscles is prevented by continued new formation. This, which suffices in health to maintain a uniform standard, may prove insufficient in disease, or it may fail entirely. The ensuing corpuscular deficiency is spoken of as anemia, a term that properly comprises a lessening of the total volume of the blood or a deficiency of any one of its constituent elements, corpuscles, hemoglobin, or albuminous and other constituents of the plasma. Changes in the plasma, however, are ill understood, and while in some cases they probably are of prime importance, clinically our attention is practically always directed to the corpuscles and the hemoglobin.

Anemias are commonly divided into the primary and the secondary anemias. Strictly speaking all anemias are secondary, that is, the consequence of some underlying, though often overlooked, or unknown, cause. Since, however, the cause of some anemias cannot be ascertained, we may continue provisionally the designation primary, essential, or idiopathic anemia. These comprise chlorosis and pernicious anemia. Splenic anemia, leukemia, and Hodgkin's disease, sometimes here

included, are not in any sense primary anemias. Secondary or symptomatic anemias, those due to obvious causes, are further divided, according to their course, into acute and chronic.

Anemia may be brought about by: (1) Hemorrhage; (2) lessened corpuscular production (deficient hemogenesis); or (3) increased destruction (increased hemolysis); that is, by the loss of a considerable volume of blood or by causes that injure either the hemopoietic tissues or the corpuscles after they have been formed. Although deficient hemogenesis is perhaps at the basis of chlorosis, the majority of cases of anemia result from increased hemolysis, to which, in cases of long-continued or very severe anemia, defective and deficient hemogenesis may be added. The hemolysis may be induced by: (1) Bacterial toxins; (2) chemical poisons; (3) cellular and other hemolysins; (4) the action of heat (burns) and of cold (paroxysmal hemoglobinemia)—which either kill the erythrocytes outright or injure the enveloping elastic membrane and permit the diffusion of the contained hemoglobin into the plasma (hemoglobinemia). Similar results may ensue from changes in the protein composition of the plasma, and from changes in the molecular concentration (electrolytic conductivity by means of ions) of the plasma and consequent changes in osmotic tension. A plasma too rich or too poor in salts (ions) leads to shrinkage or swelling of the erythrocytes and loss of hemoglobin. The hemoglobin set free in the plasma is, as a rule, speedily removed by the spleen and liver, but in some cases (occasionally in malaria, paroxysmal hemoglobinemia, etc.), the hemolysis occurs so rapidly and to such an excessive degree that the liver, spleen, bone marrow, etc., are unable successfully to cope with it and hemoglobinuria results. The hemoglobin may become converted into methemoglobin, either in the cells or in the plasma. The consequences of anemia are obvious—reduction in the oxygen-carrying capacity of the blood and insufficient nutritive supply to the organs, particularly the brain and the heart. The factors inducing the anemia may or may not implicate the leukoblastic tissues, so that the white corpuscles may remain normal, or they may become increased (leukocytosis), or diminished (leukopenia).

Following anemia—whatever its cause—regeneration of the corpuscles usually begins immediately, and it proceeds, sometimes slowly, sometimes remarkably rapidly—the rate depending apparently upon the nature of the stimulus and the vitality of the erythroblastic and leukoblastic tissues. Whatever the nature of the stimulus (infectious, toxic, chemotactic), it results at first in the discharge into the circulation of the already formed and warehoused mature cells—normal erythrocytes, polynuclear leukocytes, etc. Should these not suffice to restore and maintain the normal balance, more or less immature cells appear in the circulation—erythrocytes variable but rather small in size, distorted in shape, poor in hemoglobin, some, perhaps, nucleated and exhibiting polychromatophilia (evidence likely of immaturity rather than senility), and leukocytic antecedents of the polynuclear neutrophiles. The appearance of such cells in the circulating blood, then, is to be interpreted as an

effort on the part of the bone marrow to supply a deficiency. Under these circumstances the erythroblastic (red) centers in the bone marrow have become hyperplastic, have encroached upon and replaced more or less of the fatty (yellow) marrow of the long bones. In the more severe types of anemia, represented by the several varieties of pernicious anemia, in addition, to the foregoing, megalocytes and megaloblasts appear in the circulation in considerable number, and the bone marrow reverts to the fœtal or juvenile type (megaloblastic degeneration, Ehrlich); that is, although red, as in milder anemias, it contains many large cells—megalocytes and megaloblasts. This is interpreted as evidence of overstrain and consequent inefficient effort to replace lost cells—the production of cells of the fœtal life (metaplasia of the erythroblastic tissue). In some cases the bone marrow ultimately becomes incapable of supplying the demand; it may become exhausted, or its activity may be inhibited by the initiating infectious, toxic, or other cause, and complete cessation of its function may ensue (aplastic anemia): the bone marrow becomes pale, yellowish, or fatty throughout. In some cases the stimulus to activity involves the leukoblastic rather than the erythroblastic tissues, and leukocytosis, leukemia, etc., result. In other cases inhibition of leukoblastic activity and leukopenia ensue, as in some anemias, typhoid fever, cachexias, intoxications, etc. In leukemia and occasionally in other conditions (infections), the entire hemopoietic system (bone marrow, spleen, lymph nodes, even the liver) may revert to the fœtal type, and cells indistinguishable from bone-marrow cells may be formed in the spleen, lymph nodes, etc. Many of these cells partake of the undifferentiated, perhaps ancestral type. In lymphatic leukemia the normal granular series of bone-marrow cells may entirely disappear. In some cases the stimulus to activity is not specific, at least, not in its entirety, and both the erythroblastic and the leukoblastic tissues may be involved, as is exemplified in the occurrence of cells of the leukoblastic series in some cases of severe anemia (ordinary pernicious anemia, leukanemia), and the marked reduction, if not total absence, of the leukocytic (granular, bone-marrow) cells in cases of aplastic anemia, etc.

Plethora and Polycythemia.—An increase in the total volume of blood (so-called plethora) was formerly much believed in, but its existence is, at least, doubtful. One may suspect such a condition in a person, usually a man, who eats too much and drinks too much (especially alcohol), who is large in muscle and round in fat, who exhibits general cutaneous hyperemia and dilated venules of the face, and whose heart perhaps is large and arteries full and bounding. A serous plethora has been found to be common in chlorosis. More commonly there is an increased number of erythrocytes in a measured amount of blood (polycythemia): physiologically in the newborn, and at high altitudes in consequence of lowered atmospheric pressure and consequent increased peripheral distribution; and pathologically, in congenital heart disease, in acquired heart and pulmonary disease attended with blood stasis, in erythremia

with splenomegaly, and in cholera and other diseases in which the blood may become inspissated.

Leukocytosis.—At birth the number of leukocytes varies from about 14,000 to 19,000 per cubic millimeter; gradually the number diminishes, so that in adult life they average about 7500, the normal limit being usually set down as from 6000 to 10,000. They vary somewhat in the same individual from time to time. Under divers circumstances the number may be increased beyond the normal limits, a condition spoken of as leukocytosis—which usually is temporary, but may last for some considerable time (subacute or chronic leukocytosis). In the short-duration cases the increased number of leukocytes is derived mainly from already formed cells resting or awaiting a summons in the sinuses of the bone marrow and the capillaries of the liver and lung; but in the long-duration cases new formation occurs and the bone marrow exhibits leukoblastic changes. Leukocytosis may be physiological or pathological.

Physiological Leukocytosis.—An increased number of leukocytes is normal—at and for some time after birth; during the latter part of pregnancy and parturition; after a cold bath, exercise, and massage; and during digestion, particularly of a meal rich in protein. It is doubtful whether under any of these circumstances any new formation occurs; probably there is only a redistribution of leukocytes already formed. The increase is scarcely if ever more than one-third of the normal, and the relative proportion of the different varieties of leukocytes remains unchanged.

Pathological leukocytosis may be due to: (1) Infections, especially those associated with an inflammatory, purulent exudation. In these cases it is attributed to chemotactic influences, and there is probably a new formation of leukocytes. The absence of leukocytosis in some infections is notable—typhoid fever, malaria, measles, rötheln, influenza, mumps, early stages of tuberculosis (except meningeal form), and leprosy. (2) Zoöparasitic invasions, especially trichinosis, uncinariasis, filariasis, echinococcosis, etc. (3) Intoxications, such as illuminating gas poisoning, uremia, etc. (4) The cachexias of malignant disease and other marantic states. (5) Hemorrhage. (6) Terminal or preagonal states. (7) Certain drugs. The number of leukocytes usually varies between 15,000 and 30,000; occasionally it may reach upward of 50,000, and very rarely even upward of 100,000. Although usually there is an increase of all forms of leukocytes, there is, in contrast to physiological leukocytosis, usually also a disproportionate or so-called relative increase in some one of the types, so that one speaks of polynuclear leukocytosis, large mononuclear leukocytosis, lymphocytosis (lymphemia), eosinophilia, basophilia, and myelocytosis (myelemia). Myelocytosis aside from myeloid leukemia is a rare occurrence; but a small percentage of myelocytes is occasionally seen in certain infections and more or less severe anemias, especially in children. Basophilia also is a rare and ill-understood condition; it occurs in myeloid leukemia and rarely in certain parasitic infections. Eosinophilia (5 to 30 per cent. of the total number of leukocytes) is a significant

diagnostic sign of parasitic infections (trichinosis, uncinariasis, filariasis, ecchinococcosis, bilharziosis, etc.); it occurs also in certain skin disorders (urticaria, pemphigus, psoriasis, pellagra, purpura, etc.), in bronchial asthma, in myelocytic leukemia, in certain inflammatory exudates, etc. Lymphocytosis (a relative and absolute increase in the number of circulating lymphocytes) is normal in the infant; it is especially conspicuous in pertussis, and sometimes occurs in syphilis and certain forms of septicopyemia attended with lymph-node involvement; and it is the characteristic feature of lymphocytic leukemia. A large mononuclear leukocytosis (relative) sometimes occurs in malaria. The polynuclear neutrophilic leukocytosis is the most common and most important form; it occurs especially in infectious inflammations with or without suppuration. Significance attaches not only to an increase in the total number of leukocytes, but also to a relative increase (80 to 95 per cent.) of the polynuclear neutrophiles, even when the total number of leukocytes is only slightly above the normal. The following opinions seem warranted: (1) An early and marked polynuclear leukocytosis is usually significant of good resistance to a more or less severe infection; (2) persistence or increase in the leukocytosis is significant of continued good resistance; (3) a slight total leukocytosis, or a relative polynuclear neutrophilic leukocytosis, may mean only a minor infection, or a severe, perhaps fulminating infection, against which the forces of resistance are more or less incapable of contending; (4) a delayed and then, perhaps, only moderate leukocytosis usually means a more or less severe infection and deficient resistance; (5) a high leukocytosis with increasing toxic manifestations, while signifying good resistance, does not preclude the possibility of ultimate exhaustion of all the body forces, including the powers of resistance as represented in part by the leukocytosis; (6) a moderate or high relative (as well as absolute) polynuclear leukocytosis, while often significant of suppuration, is more properly indicative of severity of infection which may or may not lead to suppurative exudation, depending upon the associated phenomena; the diagnosis of suppuration should be made upon all the clinical evidence, and not upon one sign (the leukocytosis) alone.

ACUTE SECONDARY ANEMIA.

Etiology.—Acute secondary anemia is almost always due to hemorrhage, either external or internal, spontaneous or traumatic; in degree it usually corresponds with the rapidity and the total volume of blood lost. In rare cases acute anemia results from bacterial infection or intoxication, though these usually lead to subacute or chronic anemia. Infectious diseases, such as typhus, malaria, and typhoid fevers, and certain forms of sepsis, especially puerperal sepsis, and poisoning by nitrobenzol and other substances that rapidly destroy the blood (hemolysis), sometimes result in very acute anemia.

Pathology.—After death from very severe hemorrhages one usually finds fatty degeneration of the viscera, especially the heart muscle, the liver, the kidneys, and the stomach, and sometimes hyperplasia of the erythroblastic tissue of the bone marrow.

Symptoms.—The symptoms of acute anemia consist of pallor of the skin and visible mucous membranes; increased frequency of the heart beat, anemic murmurs, and smallness and emptiness of the pulse; coolness of the extremities, and generally low body temperature; marked dyspnœa in consequence of the marked diminution in the oxygen-carrying constituent of the blood; thirst; evidences of cerebral anemia, such as faintness, spots before the eyes, noises in the ears, vertigo, nausea, vomiting, fibrillary tremors, and perhaps convulsions (in extreme cases). The amount of blood lost in some cases is extreme (rupture of an aneurysm, ectopic pregnancy, etc.), and may lead immediately to death from acute asphyxia and anemia of the brain; but many patients survive very great and sudden losses—perhaps one-half of the total volume of blood. If not sudden, the loss of blood may be even greater, and the patient survive—repeated hematemesis, hemorrhages from duodenal or typhoid ulceration, etc.

Examination of the blood reveals oligocythemia, and usually a disproportionate oligochromemia, so that the color index is usually low—0.8 or lower. The oligocythemia rarely reaches the high grade observed in pernicious anemia; thus, after repeated hemorrhages from gastric ulcer, the count may be scarcely less than 2,000,000 or 1,800,000. Regeneration of the blood begins almost immediately, and continues apace, so that in some cases of severe hemorrhage restitution is almost complete at the end of ten to fourteen days; usually, however, the normal condition is not reached for several weeks or months. The hemoglobin value is restored less rapidly than the corpuscles, and may remain relatively low (low color index) for a considerable time. The total volume of the blood is usually soon restored by absorption of fluids (as well as salts) from the lymphatics and elsewhere.

Microscopically, anisocytosis may be observed, and while a few erythrocytes may be undersized, the majority appear swollen after severe hemorrhages; poikilocytosis is usually slight; polychromatophilia is often present early, but soon disappears; nucleated corpuscles (mostly normoblasts) appear early, sometimes in large numbers and intermittently, and may be found until restitution of the blood is complete. Significant of regeneration, the absence of erythroblasts augurs ill. The leukocytes are often slightly increased—polynuclear neutrophilic leukocytosis, which usually disappears with convalescence. Rarely slight lymphocytosis or myelocytosis has been observed. In severe cases the blood approaches that of pernicious anemia—marked oligocythemia, high color index, marked poikilocytosis, megalocytes, etc.

Prognosis.—The prognosis varies with the amount of blood lost, the rapidity of the loss, the general condition of the patient at the time of the hemorrhage, and the evidences of regeneration.

Treatment.—The treatment should consist of rest in bed, with depression of the head; ligation of the bleeding vessel, if possible; and the injection into the veins of a volume of physiological saline solution sufficient to maintain the circulation (500 to 2000 c.c.). Sterile 0.9 per cent. sodium chloride solution is commonly used, but a preferable solution (which must be sterilized) is the following:

℞—Sodium chloride	9 0
Potassium chloride	0 1
Calcium chloride	0 25
Water	1000 0

—Hare.

In sudden severe hemorrhages likely to prove immediately fatal, Crile advises resort to rapid rhythmic pressure upon the chest with a view to produce artificial respiration and a moderate artificial circulation; at the same time, by means of a cannula inserted in a large artery near the heart, he injects some physiological saline (or Ringer's or Locke's) solution, and as soon as the fluid has begun to flow, by means of a hypodermic needle inserted into the rubber tubing near the cannula, he injects 15 to 30 minims (1 to 2 c.c.) of adrenalin chloride, 1 to 1000; the injection may be repeated in a minute, if required. This brings about a rising arterial and coronary pressure, and stimulates the heart to renewed activity. Crile believes this treatment more effective than the use of stimulants, nitroglycerin, intravenous infusions, electricity, needling of the heart, or direct cardiac massage. The later treatment is that of the primary disease; and in addition, measures, to be mentioned under chronic anemia, designed to restore the blood to the normal.

CHRONIC SECONDARY ANEMIA.

Etiology.—Chronic secondary anemia may be due to: (1) Bacterial infections, whence it is a concomitant of all infectious diseases, especially typhoid fever, acute rheumatic fever, septicemia, diphtheria, syphilis, tuberculosis, etc. The anemia of the acute infectious diseases, however, is often subacute rather than chronic, and, as a rule, responds promptly to treatment. (2) Zoöparasitic infections, such as malaria and the different worms (Uncinaria americana, Dibothriocephalus latus, Filaria bancrofti, Schistosoma hæmatobium or japonicum), which often produce an anemia scarcely distinguishable clinically from progressive pernicious anemia. (3) Intoxications, (*a*) biological (not separable from the infectious diseases); (*b*) inorganic, such as lead, arsenic, mercury, phosphorus, potassium, certain coal tar derivatives, etc.; (*c*) autogenic poisons, such as occur in disorders of the gastro-intestinal tract, nephritis, carcinoma, so-called cholemia, pregnancy, etc. (4) Prolonged drains upon the system, with loss of the albuminous constituents of the blood, such as occur in prolonged suppuration, nephritis, prolonged lactation, dysentery, etc. (5) Repeated hemorrhages—gastric and

duodenal ulcer, liver cirrhosis with hemorrhages, hemorrhoids, menorrhagia, metrorrhagia, hemophilia, etc. (6) Inanition and starvation, due to deficient food or inability to swallow food, such as occur in œsophageal carcinoma and somewhat similarly in obstruction of the thoracic duct. (7) Insanity. In these latter cases the plasma often suffers much, and on account of inspissation the blood corpuscles may be relatively numerous (5,000,000 or more to the cubic millimeter).

Symptoms.—In many cases the symptoms due to the anemia are intimately bound up with those due to the primary disorder, and it is difficult to separate the one from the other. In general, however, the symptoms of the anemia are those of general nervous and muscular irritability and weakness. The patient is readily fatigued by bodily or mental exertion, he is incapable of concentrated or continued mental effort, and headache, vertigo, syncopal attacks, tinnitus, dimness of vision, insomnia (or somnolence), are common, and occasionally nausea and vomiting may be present. The appetite is poor, the digestion often imperfect (variability of HCl) and often painful (hyperesthesia of the gastric mucosa), and constipation is the rule (defective intestinal peristalsis). The heart's action is weak, though readily excited—whence palpitation and arrhythmia, as well as dyspnœa, are common; a systolic murmur is usually present, and a venous hum (*bruit de diable*) may be heard in the neck; the second pulmonary sound is accentuated; from dilatation, a relative mitral insufficiency may occur; the pulse is small, weak, and compressible, and the extremities are usually cool. Slight œdema, due to increased permeability of the vessels, occurs in some cases, and hemorrhages into the mucous membranes, under the skin, and in the retina and cerebral meninges are not uncommon. The urine is often increased in amount. Occasionally there is slight fever (anemic fever). There may be amenorrhœa or menorrhagia; and perhaps impotence.

The blood reveals oligocythemia (4,000,000 to 2,000,000), and disproportionate oligochromemia (65 to 30 per cent.); therefore a low color index (0.8 to 0.6). In some cases, however, these figures are exceeded, and approach those of pernicious anemia. Microscopically, the corpuscles are pale, and reveal anisocytosis, poikilocytosis, and polychromatophilia; nucleated corpuscles (usually normoblasts) may be present. There is little, if any, leukocytosis due to the anemia as such, but leukocytosis as a manifestation of the primary disorder is not uncommon.

Diagnosis.—The diagnosis has to do not only with the recognition of the anemia, but also with its degree, and largely with its underlying disorder. Cases of so-called simple anemia are usually chlorosis, pernicious anemia, tuberculosis, gastric carcinoma, neurasthenia, etc., and an effort should be made to differentiate these.

Prognosis.—The prognosis is that of the primary causative factor.

Treatment.—The treatment is primarily concerned with the detection and removal of the underlying cause. In addition, however, the patient's recovery may be materially aided by attention to the diet, and by suitable medicines. The diet should be varied and nutritious, and should

comprise a generous amount of red meats, milk, eggs, as well as other iron-containing articles, especially vegetables. Of medicaments, iron is of prime importance, and should be given in all cases. As a rule, no preparation is better than Blaud's pills, which should be given in generous doses (10 to 15 grains, 0.65 to 1 gram, three times a day); in some cases reduced iron, the dried sulphate or the citrate of iron, or Basham's mixture render excellent service. Arsenic is often a useful adjuvant, and may be given as Fowler's solution, 2 minims (0.12 c.c.), three times a day. Occasionally bitter tonics and hydrochloric acid are indicated to improve the appetite, but hydrochloric acid had better be withheld unless we are certain that there is a deficiency of hydrochloric acid in the gastric juice. The following is often productive of excellent results:

℞—Reduced iron	80 grains	5 2
Quinine sulphate	20 grains	1 3
Strychnine sulphate	2 grains	0 13
Arsenous acid	1 grain	0 06

Mix and make 40 pills.
S.—One pill three times daily.

Minute doses of mercuric bichloride are sometimes serviceable, even in non-syphilitic cases. The debility of exhausting disorders, such as typhoid fever, prolonged lactation, neurasthenia, etc., is often much improved by the lactophosphates, the hypophosphites, cod liver oil, etc. In cases that do not respond promptly iron may be given hypodermicly, the citrate in doses of ½ grain (0.03 gram) to children, 2 grains (0.13 gram) to adults, every other day. The bowels should be kept open once daily. Other suitable measures consist of medicated baths, massage, and change of climate—high altitudes being of undoubted service in some cases, since it seems that the increase in the corpuscular count obtained at high altitudes does not always disappear upon return to lower levels.

CHLOROSIS.

(Chloranemia; Green Sickness.)

Chlorosis is a form of anemia occurring in young girls about the time of puberty, and characterized by marked oligochromemia and sometimes by a peculiar greenish tint of the skin.

Etiology.—Chlorosis is peculiarly a disease of the female sex, occurring in blondes more frequently than in brunettes, and about the age of puberty, that is, from the fourteenth to the seventeenth year, but recurrences up to and probably beyond the twenty-fifth year are common. True chlorosis never occurs in males. It is most common among domestic servants and immigrants, particularly Irish; but if many chlorotic girls are underfed and overworked, have insufficient exercise and fresh air, many have been reared under the most favorable circumstances and do no work; if many have been subject to distressing emotional disturbances, such as worry, youthful love affairs, etc., others are unrestrained and

free from care. The mothers of some chlorotic girls themselves were chlorotic about the age of puberty.

The exciting cause of the disease is unknown—despite many hypotheses. Virchow attributed it to hypoplasia of the cardiovascular system, especially the aorta, and the genitalia; but these, though sometimes observed, can scarcely cause the disorder, since it is difficult to comprehend the relationship between hypoplasia of the aorta and oligochromemia, since such hypoplasia does not cause chlorosis in boys, and whereas iron may cure chlorosis, it must be powerless to correct the hypoplasia. Sir Andrew Clark believed the disease to be a manifestation of intestinal auto-intoxication due to coprostasis—a view with little foundation in fact, since otherwise most girls and women would be chlorotic, and because evidences of intestinal putrefaction have not been found constantly in the urine of chlorotics. The occurrence of chlorosis in girls about the time of puberty suggests an intimate relationship between the series of changes then taking place and the alterations in the blood; possibly time may show that some derangement in the internal secretion of the ovary developing at the time the ovary begins to functionate actively, may be answerable for the disease. The majority of cases cannot be incipient pulmonary tuberculosis, as has been suggested, since they are readily cured with iron; nor is it likely that they are cases of more or less ill-declared hyperthyroidism, associated or not with tuberculous lesions in the thyroid. Unquestionably chlorosis seems to be much less common than it was years ago, which is due to the fact that the causes of many chloranemic conditions are more commonly recognized; this suggests the additional fact that chlorosis in reality is a secondary anemia, of which the cause is not always demonstrable. The common absence of marked changes in the erythrocytes, the deficiency of pigments in the urine and feces, and the non-occurrence of even slight jaundice, suggest that the disease is due to defective hemogenesis rather than augmented hemolysis.

Symptoms.—The symptoms, for the most part, are those of the concomitant anemia, and as such do not differ from those described in connection with chronic secondary anemia. The early symptoms are usually breathlessness, palpitation of the heart (which too often suggests heart disease), vertigo, faintness, weariness of the mind and body without obvious cause, and indisposition to exertion. In marked contrast with the subjective complaints, the girl often presents a much prized though deceptive peach complexion (chlorosis rubra), especially upon exertion or excitement, and she is more likely to be fat and round than thin and angular. In marked cases, however, the skin exhibits the ordinary pallor of any secondary anemia; occasionally it presents a peculiar yellow-greenish color, most notable on the face, ears, dorsal surface of the hands, and the chest; pigment spots especially about the joints are often present; the eyes are bright, the conjunctivæ transparent, and the sclerotics bluish. Indeed, the observing physician may usually make the diagnosis from mere inspection, and yet the disease is frequently overlooked. The

appetite is often characteristically capricious, the patient evincing a liking for most extraordinary articles, such as chalk, paper, pickles, etc. (pica chlorotica); epigastric distress is common after eating, and constipation is the rule. Appropriate examination often reveals gastric hyperacidity, frequently gastroptosis and even gastrectasis (especially if the patient has succumbed to the vanity of tight lacing). Examination of the heart, suggested by the palpitation, usually reveals excited action; a systolic murmur, commonly with its point of maximum intensity toward the pulmonary area, though frequently audible over the entire precordium; a venous hum in the neck (*bruit de diable*); and often percussion evidence of enlargement (dilatation) of the heart. The murmur is usually attributed to anemia (and partakes of the characteristics of so-called anemic, functional, murmurs); in some cases, however, it is probably due to dilatation of the root of the pulmonary artery, and in some cases to relative mitral insufficiency (irregular action of the papillary muscles). Venous thrombosis (femoral, cerebral sinuses) is by no means uncommon (2 per cent. or more of the cases). The extremities are often cold (defective circulation). Œdema or puffiness of the feet and ankles is sometimes present, and has been mistaken for evidence of nephritis or heart disease. Divers nervous manifestations are common—headache, neuralgic pains in the neck and the back, and reflected elsewhere, general irritability and capriciousness, vulgar "hysterics," dermographism and other evidences of vasomotor instability (or ataxia), etc. Slight fever occurs occasionally.

The total volume of the blood is increased, due to increase of the plasma (serous plethora; polyplasmia), in consequence of which the vascular system is overdistended. The blood as it flows from the puncture is pale, but the characteristic feature is the marked reduction in the hemoglobin. The erythrocytes are often normal in number; they are usually over 4,000,000, that is, about 80 per cent. of the normal, whereas the hemoglobin varies from 35 to 65 per cent. in different cases, averaging about 40 per cent. The color index thus approaches 0.5. In some cases, however, there is quite a reduction in the erythrocytes—to 3,000,000 or 2,500,000, or even less; but the disproportionate reduction of the hemoglobin (the low color index) is a constant feature. Microscopically the individual erythrocytes are pale (low color index, deficiency of hemoglobin), and anisocytosis (small cells) and poikilocytosis may be present and sometimes are marked (severe cases); large erythrocytes (megalocytes), however, are rarely if ever seen; nucleated red cells are sometimes present; polychromatophilia is rare. The leukocytes are usually normal in number, but a slight increase especially of the lymphocytes is by no means unknown.

Diagnosis.—As already stated, the diagnosis is often suggested by mere inspection of the patient, but the frequency with which the disease is overlooked and mistaken for something else is deplorable. If a hemoglobinometer is not at hand, recourse may be had to the simple device of comparing a drop of the patient's blood with a drop of presumably

normal blood, whereupon the deficiency of hemoglobin is immediately apparent. Care should be taken not to mistake beginning pulmonary tuberculosis for chlorosis, nor chlorosis for nephritis (on account of slight œdema), or heart disease (on account of palpitation and a murmur), or hyperthyroidism, or neurasthenia. The examination of the blood is usually conclusive, but one must bear in mind that a chloranemic condition of the blood is the unusual condition in secondary anemias.

Prognosis.—The prognosis is good. Recovery, under proper treatment, ensues in from two to four or six months, but relapses are not uncommon, largely because of too early discontinuance of treatment.

Treatment.—Rest in bed with an abundance of fresh air and sunlight, for a time at least, is advisable in all but the mildest cases. The food should be nutritious and generous in amount—milk, eggs, meats, fish, stewed fruits, green vegetables, etc.; but it is sometimes a tax on the patience of the physician to convince a chlorotic girl that she can digest food other than chalk and pickles, and that meat in particular will agree with her delicate stomach. Iron is a specific remedy. It is a matter of comparative indifference which preparation is given, provided the iron is given in generous amount. No preparation is better than Blaud's pills:

℞—Dried iron sulphate,
Potassium carbonate, of each . . . . . 100 grains. 6.6
Mix and make 40 pills.
S.—One pill three times a day for the first week, two pills three times a day for the second week, and three pills three times a day thereafter.

The treatment must be persisted in for at least three months, despite the conviction of the patient that she is cured earlier, since otherwise recurrence is almost inevitable. Under the influence of iron the erythrocytes soon reach the normal number, and while the hemoglobin value increases, considerable time is often required to attain the normal percentage. Exactly how the iron acts is a matter of speculation, and the theoretical objection to large doses, whereby more iron is given within a few days than is contained normally in the entire body, is not sustained by any clinical or experimental facts. Indeed, a markedly chlorotic patient, despite the evident need of iron in her blood, excretes iron in her feces, even before the administration of iron is commenced. Those who prefer them may use reduced iron, the citrate of iron and potassium, or the citrate, the dried sulphate, the pyrophosphate, the lactate, or the succinate of iron or the tincture of the chloride; one or the other of these may be given from time to time should convalescence prove tedious, in which case also minute doses of arsenic or of manganese may prove serviceable. The so-called organic preparations of iron, said to reproduce the normal iron of the body, are in no way superior, and in many ways inferior, to the preparations mentioned.

In addition to the foregoing, the patient's bowels should be kept open daily—for which purpose, as a rule, nothing is better than the compound rhubarb pill (U. S. P.):

℞—Rhubarb		48 grains	3 20
Purified aloes		36 grains	2 40
Myrrh		12 grains	0 80
Oil of peppermint		4 minims	0 25

Mix and make 48 pills.

S.—One or two pills night and morning.

Cascara sagrada, saline cathartics, phenolphthalein (1 to 2 grains, 0.06 to 0.13 gram), etc., are also efficacious. Cleansing of the bowel should precede the beginning of the iron treatment. To patients with evident intestinal fermentation, it is wise to give for a time so-called intestinal antiseptics, especially bismuth salicylate, β-naphthol, thymol, aspirin, preparations of the lactic acid bacillus, etc. In other patients with imperfect digestion, hydrochloric acid, nux vomica, and the bitter tonics prove efficacious; but one must remember that hyperchlorhydria is common in chlorosis.

PERNICIOUS ANEMIA.

Pernicious anemia is a chronic disease of the hemopoietic tissues characterized by hemolysis of unknown cause, abnormal hemogenesis, and alternating periods of remission and relapse.

There has been and there still is much discussion as to what is and what is not properly pernicious anemia. There are several disorders that may give rise to clinical and blood pictures resembling, if not quite identical with, pernicious anemia that should be excluded, since they are obviously secondary anemias of severe (perhaps one might say also pernicious) and even fatal type. These include: (1) Some cases due to severe and frequently repeated hemorrhages; (2) certain cases of severe malarial, syphilitic, and other infections; (3) Dibothriocephalus latus and other zoöparasitic infections, in which the blood picture may be quite identical with that of pernicious anemia and the termination fatal; (4) cases of severe and fatal anemia occurring during pregnancy or soon after parturition, and due presumably, as is the vomiting, nephritis, and eclampsia of pregnancy, to some auto-intoxication, or to puerperal hemorrhage or infection; these occur, as a rule, in young subjects (thirty to thirty-five years), do not show the remissions characteristic of pernicious anemia, and are often curable upon removal of the initiating cause; (5) cases due to primary changes in the bone marrow, such as primary or secondary tumor formation, or leukemia (so-called myelophthisic anemia). There remains a series of cases of unknown cause to which the term pernicious, idiopathic, essential, Addison's, or Biermer's anemia may be applied.

Etiology.—Pernicious anemia is especially common in adult life: more than half the cases occur after the fortieth year; it is rare in childhood. It is more than twice as common in men as in women (eliminating the cases occurring in pregnancy). The cause of the disease is not known. It has been variously attributed to nervous shock; to repeated or long-

continued, perhaps concealed, gastro-intestinal hemorrhages (which are more likely a result than a cause of the disease); to atrophy of the gastro-intestinal mucosa (which, however, is not always present, and if present may as well be a result as a cause, or a manifestation of the same cause); to subinfection with intestinal bacteria; and, especially by Hunter, to oral and gastro-intestinal sepsis. Hunter states that the disease is a well characterized specific infective disease of hemolytic nature localized in the alimentary tract; that in its etiology long-standing sepsis, oral and gastric, plays an essential and important antecedent and concurrent part; that anemia is but one of the symptoms, of which there are three others: (1) Glossitic and gastro-intestinal; (2) hemolytic; and (3) febrile and nervous—far more characteristic and due not to the anemia, but to the infective agencies underlying the disease. Disapproving of the term pernicious, he separates this so-called Addisonian (primary cryptogenetic) anemia, a specific infective disease, sharply from other types of severe or pernicious (Biermer's) anemia, which he believes may be due to other types of general sepsis; and all these types, which he characterizes as infective anemias, he differentiates from so-called non-infective anemias—that is, chlorosis and all the ordinary posthemorrhagic and other types of secondary anemia.

Pathology.—The conspicuous lesions consist of pallor, a lemon tint, or even slight jaundice of the skin; pallor of all the viscera; a peculiar bright red color of the muscles and light yellowish fat; fatty degeneration of the liver, heart, and kidneys (toxic, perhaps, rather than anemic degeneration); petechial hemorrhages in the subcutaneous, submucous, and subserous tissues; effusions in the serous sacs; systemic or disseminated degeneration of the posterior columns, and sometimes of the lateral columns and of the gray matter of the spinal cord (perhaps also toxic rather than anemic). Hunter emphasizes the importance of certain so-called infectious lesions; a peculiar glossitis, gastritis or gastric atrophy, and patchy enteritis, which he believes to be insufficient in themselves to originate the intense anemia by an interference with nutrition. The liver, spleen, lymph nodes, and hemolymph nodes usually show considerable deposit of blood pigment and large numbers of phagocytes (macrophages) containing more or less completely destroyed erythrocytes—all evidences of excessive hemolysis. The fatty portions of the bone marrow are usually more or less replaced by red marrow resembling the fœtal or juvenile type (megaloblastic degeneration or metaplasia), interpretable as an effort to compensate for the excessive hemolysis; there may be also some evidences of leukoblastic hyperplasia. In some cases the bone marrow is either incapable of responding or ultimately becomes exhausted, whereupon it may be more or less completely yellow (fatty) throughout (aplastic anemia).

Symptoms.—The disease, as a rule, is exceedingly insidious in onset, and it often has made considerable progress, that is, the blood is remarkably bad, when the patient first seeks medical advice. The initial manifestations are variable and permit of some grouping of the cases: (1)

Gradually increasing weakness, especially upon exertion, with dyspnœa and palpitation of the heart; (2) gastro-intestinal derangements—sore mouth, anorexia, nausea, vomiting, diarrhœa, or perhaps constipation; (3) general nervous irritability or inadequacy, lost power of mental effort or concentration, headache, vertigo, tinnitus, fainting, somnolence, cutaneous parasthesias and ataxia, spasticity of the muscles of the legs, etc. In the course of time the symptoms become aggravated. The weakness and weariness increase; mental effort and concentration are even less well sustained than previously; and dyspnœa and palpitation of the heart become more marked. Hemorrhages (usually petechial) may occur into the skin, visible mucous membranes, retina, etc.; nausea and vomiting may become aggravated, and are often associated with paroxysmal attacks of epigastric pain and diarrhœa. In a majority of the cases nervous phenomena become more or less obtrusive, such as various paresthesias, numbness, tingling, etc. (even in the absence of demonstrable changes in the spinal cord); in other cases symptoms suggestive of tabes dorsalis (severe pains, absent knee-jerks, etc.) occur; they may even precede and ultimately become more marked than symptoms referable directly to the anemia; in other cases, spasticity of the muscles of the legs and increased knee-jerks, associated late, perhaps, with incontinence of urine and feces, supervene. To a not uncommon mental apathy a depressive psychosis (melancholia) sometimes succeeds.

On examination the patient may present a general cutaneous pallor, such as might be encountered in any severe anemia; in other cases there is a characteristic waxy, lemon-yellow color of the skin; in some cases there is even slight jaundice (not as frequent as is often diagnosticated); and rarely there is more or less pigmentation (and perhaps associated leukoderma), suggesting Addison's disease. The subcutaneous fat is usually well preserved, and there is often comparatively little loss of weight. As a rule, there is a notable absence of abnormal physical signs. The heart is usually somewhat dilated; there may be hemic murmurs over the heart and in the cervical veins, or a murmur of relative mitral insufficiency; the pulse may be full but not well sustained; the blood pressure is usually low (often less than 100 mm. Hg., systolic or maximum); and a capillary pulse may be visible. Œdema of the legs and hands is common, and transudations may occur into the pleura, peritoneum, etc. The spleen and liver may be slightly enlarged; occasionally also the palpable lymph nodes. Hydrochloric acid and the digestive ferments are usually absent or greatly diminished; there is rarely any marked dilatation of the stomach or motor insufficiency except perhaps at the time of the paroxysmal pains. The urine is usually pale and of low specific gravity; in from one-fourth to one-half of the cases it contains a small amount of albumin and perhaps a few casts; occasionally it is dark in color, due usually to an excess of urobilin. Fever occurs in about three-fourths of the cases—often (100° to 102°) for a week or two at a time.

The blood is abnormally fluid, and the total volume is lessened. The red cells are remarkably reduced, numbering often 2,000,000 or less

when the patient first consults a physician—which in itself suggests the disease; later they may fall to 1,000,000 or less (even 143,000, Quincke). The color index is high, 1.0 to 1.5 or more (often increasing with the progress of the disease and lessening during the periods of remission); this is characteristic of the disease, and attributable not to hemoglobinemia (which, however, is occasionally present), but to the large number of macrocytes rich in hemoglobin. Poikilocytosis is usually extreme; there is marked anisocytosis, megalocytes being present usually in such size and numbers as to increase the average diameter of the erythrocytes (offsetting the lessened diameter of the microcytes); polychromatophilia (diffuse and granular) is exceedingly common (more so than in any disease other than lead poisoning); and nucleated red corpuscles, especially megaloblasts, are present in more than 90 per cent. of the cases. The leukocytes are usually diminished (5000 or less)—usually a polynuclear deficit; there may be a relative, sometimes an absolute, lymphocytosis. Sometimes the leukocyte count is normal, especially during the remissions; rarely there is a slight leukocytosis (blood crisis; complicating, perhaps, terminal infection). A few myelocytes are often encountered. The blood platelets are lessened. Often what are spoken of as blood crises occur—sudden increase of the erythrocytes (50 or more per 1000 leukocytes), and of the leukocytes (especially the eosinophiles). The crises may be spoken of a normoblastic or megaloblastic, depending upon the predominating type of nucleated cell. These often are favorable signs of bone-marrow activity and are followed by general and blood improvement; but sometimes they represent the final effort of an exhausted marrow and may portend an early fatal issue.

A characteristic of the disease is alternating periods of remission and relapse—which often succeed each other with considerable regularity. As a matter of fact, when the blood is poor it is remarkably inconstant, the erythrocyte count either increasing or diminishing. A total count of over 4,000,000 is rarely attained; but the blood may remain stationary at a good, rather high level for from two to six, sometimes six to twelve, months or more. Considerable improvement as well as deterioration of the blood not infrequently occurs without noteworthy change in the general subjective complaints of the patient; these (as well as the fatty degeneration of the viscera and the lesions in the spinal cord) likely are due in part only to the anemia as such, and in part to the underlying (toxic?) factor.

Diagnosis.—The diagnosis should be made more frequently than it is, and might readily be made by attention to Addison's statement that it is a disease "occurring without any recognizable cause whatever; where there has been no previous loss of blood, no exhausting diarrhœa, no chlorosis, no purpura, no renal, splenic, glandular, strumous, or malignant disease, . . . and without any organic lesion that could properly or reasonably be assigned as an adequate cause of any serious consequences." In addition to the phenomena common to any severe anemia, the following are significant of pernicious anemia: Severe cause-

less anemia in a middle-aged or elderly male subject; generally well-preserved panniculus adiposis with little loss of weight; sore mouth; paroxysmal attacks of epigastric pain often attended with diarrhœa; retinal and other hemorrhages; extraordinarily low erythrocyte count at the first examination, that is, the patient's general good appearance is in notable contrast to the condition of his blood, and he is often able to continue at his work with an erythrocyte count approaching 1,000,000; high color index; normal leukocyte count or leukopenia; an unusually large number of megalocytes (over 30 per cent. of the total erythrocyte count being quite diagnostic), megaloblasts, and poikilocytes; polychromatophilia; and alternating periods of remission and relapse. All types of severe secondary anemia may be excluded by the detection of a causative factor, especially severe and frequently repeated hemorrhages, pregnancy, severe infections, intestinal parasitism (ova in the stools), etc. Gastric carcinoma, myxœdema, etc., sometimes bear a more or less superficial resemblance to pernicious anemia, but with ordinary care they should be readily excluded.

Aplastic anemia virtually is only an extremely severe and rapidly fatal form of pernicious anemia, characterized by slight if any response on the part of the bone marrow to the demand for erythrocytes. The marrow of the long bones is yellow throughout—aplastic (rather than hyperplastic or metaplastic) as contrasted with the ordinary type of pernicious anemia. It is a disease that especially affects young women before the thirty-fifth year; subcutaneous and submucous hemorrhages are more common; it is steadily progressive, showing none of the characteristic remissions; and it usually leads to death within a few months (three or thereabouts). The blood is characterized by the common extraordinary oligocytosis, but the color index is likely to be low (0.8); erythroblasts are usually absent and so-called erythroblastic crises do not occur; poikilocytosis, anisocytosis, and polychromatophilia are less marked than in the ordinary type of pernicious anemia; there is marked leukopenia, due largely to a loss of granular leukocytes, so that most (75 to 90 per cent.) of the white cells are lymphocytes; and the blood platelets are markedly lessened.

Leukanemia is a term given by Leube to a blood state that combines some of the features of pernicious anemia and of leukemia, especially megaloblasts, myelocytes, and lymphocytes. The majority of the cases can, with little difficulty, be classified as pernicious anemia, on the one hand, or leukemia, on the other; in the terminal stages of both of these diseases blood findings significant of the other are not rare.

Prognosis.—The disease usually, if not invariably, runs a fatal course in from one to three years; occasionally, as in aplastic anemia, within a few months; perhaps rarely it continues for five or more years with uncommonly long periods of remission. Death usually ensues from progressively increasing weakness, œdema of the lungs, or terminal infections.

Treatment.—The essentials of treatment comprise prolonged rest (mental and physical), an abundance of fresh air, and good, nutritious,

readily digestible food (especially milk and vegetables, but not too much nitrogenous food); and the administration of arsenic and hydrochloric acid. Arsenic seems to act by stimulating the erythroblastic centres in the bone marrow. It should be given cautiously, in gradually increasing doses, to the point of toleration (epigastric pain, nausea, vomiting, diarrhœa, puffiness and itching or burning of the eyelids, etc.). Arsenous acid, Fowler's solution, or sodium cacodylate, may be given. In some cases good results follow hypodermic administration: sodium cacodylate, $\frac{1}{8}$ grain (0.008 gram), iron citrate, 2 grains (0.13 gram), daily or every second day. Hydrochloric acid is indicated on account of the gastric achylia, but it is said to have also other beneficial effects. Particular attention should be paid to the teeth and mouth. The services of a dentist are often of the greatest utility. Antiseptic mouth washes should be used. Lavage of the colon also is serviceable, especially in the event of indicanuria and other evidences of supposed intestinal decomposition. Hypodermoclysis with physiological saline solution also has been commended. Cholesterin (3 per cent. solution in hot olive oil, 100 c.c. daily) has been recommended with a view to combining a hemolysin in the blood, which is believed to rob the erythrocytes of their cholesterin and lead to hemolysis, analogous to the action of cobralecithin. Large amounts of milk, cream, and butter tend to achieve the same object. Red bone marrow has fallen into disuse; by some its occasional good results have been attributed to the glycerin contained in the commonly used preparation—the glycerin extract.

LEUKEMIA.

(*Leukocythemia.*)

Leukemia is a disease of the hemopoietic system, characterized by hyperplasia of the leukoblastic tissues and by marked and usually permanent increase in the number of circulating leukocytes.

The older writers described three types of leukemia—the splenic, the lymphatic, and the medullary; but under the influences of Ehrlich these became merged into two, the splenomedullary and the lymphatic—which were held to be sharply differentiated the one from the other. The one, the lymphatic type (lymphemia), was believed to be due to primary disease of the lymph nodes; the other, the splenomedullary or myelogenous type (myelemia) was believed to be due to primary disease of the bone marrow. But with the determination of the fact that a number of cases of lymphatic leukemia occur with slight or no noteworthy changes in the lymph nodes, and that in all cases of leukemia more or less conspicuous changes occur in the bone marrow, the possibility of all cases of leukemia being myelogenous seemed proved.

The data now at hand warrant the opinion that leukemia is a disease of the leukoblastic tissues throughout the body—the bone marrow, the spleen, and the lymph nodes; that the process may begin in either the bone marrow or the lymph nodes (it is not likely that it ever begins in the

spleen); that in some cases the lesions are most marked in the bone marrow, in others in the lymph nodes, but that changes are always present in the bone marrow; that, depending perhaps upon the nature of the initiating (chemotactic?) factor, the leukoblastic proliferation in one series of the cases is of the lymphocytic or lymphoid type, and gives rise to a large number of circulating lymphocytes, whereas in another series of cases it is of the myelocytic or myeloid type, and gives rise to a large number of circulating myelocytes; and that the essence of the process is a reversion to the more or less undifferentiated leukoblastic tissue of fœtal life. This is suggested by the fact that the circulating lymphocytes and myelocytes of leukemia differ materially from the normal circulating lymphocytes and bone-marrow myelocytes (reversion to a common ancestral type?), and by the fact that the process, whether myeloid or lymphoid, soon involves in similar metaplasia tissues other than that where it originated; that is, the bone marrow may become converted into lymphoid tissue, or the lymph nodes and the spleen may become converted into myeloid tissue—these respective changes being metaplasias rather than the result of the proliferation of so-called metastases.

The foregoing facts permit of the differentiation of the cases into two main types: (1) Myelocytic or myeloid (splenomedullary), and (2) lymphocytic or lymphoid. In some respects these represent extremes of type rather than distinct diseases; some cases do not fit well into either group, and may be spoken of as atypical (transitional, intermediate, or mixed) forms.

Etiology.—The cause and the nature of the disease are not known. Many, especially the acute, cases run a course strongly suggesting an infection, but no infectious agent has yet been discovered, if one excepts Spirochæta lymphatica (White and Proescher), which still awaits confirmation. Warthin and others are inclined to look upon the disease as allied to the tumor formations, believing that transitions occur from leukemia through myeloma to ordinary sarcoma. In some cases there is a history of antecedent infections, such as malaria, syphilis, typhoid fever, tonsillitis, puerperal and other types of septicopyemia, etc.; but in most of the cases the supposed infection is in reality the declared, although not diagnosticated, leukemia.

Myelocytic or Myeloid Leukemia.—Myelocytic or myeloid leukemia, the most common form, occurs especially in males (two-thirds of the cases), and is most common in the fourth and fifth decades (60 per cent. of the cases).

Pathology.—The lesions consist of myelocytic hyperplasia of the leukoblastic tissues of the bone marrow and of myelocytic (or myeloid) metaplasia of the spleen and perhaps also of the lymph nodes. The marrow, especially of the long bones, but also of the ribs, vertebræ, sternum, etc., becomes converted into a reddish or grayish-red tissue, and the fatty marrow of the long bones gradually disappears. The spleen becomes enlarged, and may weigh 4000 to 6000 grams or more; its shape is preserved; it varies in consistency with the amount of sclerosis;

on section it usually much resembles the associated bone marrow, but it may show areas of infarction, hemorrhage, or sclerosis. The liver also becomes enlarged, and may weigh as much as the enlarged spleen, or more. The lymph nodes throughout the body (the superficial and deep chains, the hemolymph nodes, the tonsils, etc.) also may be, but are not always, enlarged. Microscopically the bone marrow and the spleen exhibit myeloid transformation—myelocytes and cells derived therefrom (all the granular leukocytes), mononuclear cells (perhaps ancestors of the myelocytes), megakaryocytes (with divers sorts of inclusions), erythrocytes and erythroblasts in various and atypical stages of development, etc. Similar changes may be found also in the lymph nodes. The capillaries of the liver, kidneys, lungs, etc., are overfilled with similar cells, and there are sometimes extra-capillary collections, and degeneration of the parenchymatous cells.

Symptoms.—The onset of the disease is insidious: the patient may seek advice on account of progressive loss of flesh and strength, the discomfort occasioned by a large mass in the region of the spleen, various gastro-intestinal disturbances, dyspnœa and palpitation of the heart, especially on exertion (and other evidences of anemia), or a sudden, perhaps serious hemorrhage. Anemia is not an early symptom, but becomes marked as the disease advances, and is attended by the usual symptoms. Hemorrhages occur in 80 per cent. of the cases: epistaxis is the most common; but frequently there is bleeding from the gums, the stomach, the intestine, the uterus, etc.; into the skin (purpuric and ecchymotic spots), in the retina (blindness), into the brain (hemiplegia, etc.), and into the serous cavities. The first hemorrhage is sometimes severe and fatal. Gastro-intestinal disturbances (anorexia, nausea, vomiting, diarrhœa, etc.) are sometimes marked, and have been found with unusual (sometimes ulcerated) lesions in the intestines. Occasionally there is more or less marked dyspnœa, due in part, perhaps to myeloid deposits in the lungs and the respiratory mucosa. Hemorrhage or myeloid nodes may cause dimness of vision or blindness if they occur in the retina or optic nerve or disk; exophthalmos, if in the orbit; deafness, or Ménière's disease, if in the semicircular canal, etc. Priapism is occasionally a distressing symptom. Fever (102° to 104°) occurs from time to time in almost if not quite all cases. Toward the end evidences of anemia often become ascendant—dyspnœa, palpitation, œdema, and other evidences of cardiac weakness and dilatation.

Examination reveals a greatly enlarged spleen—which may reach far beyond the median line and into the pelvis; it is usually hard, but sometimes painful and tender; it reveals distinct notching; often it progressively increases in size, but when very large it may remain stationary, and it may become smaller after a severe gastro-intestinal hemorrhage or diarrhœa, or in consequence of progressive sclerosis. The liver also is usually enlarged; and the superficial lymph nodes may be palpably enlarged. The bones may be tender to percussion. The urine contains a great excess of uric acid (more than in any other disease), due to

destructive katabolism of nucleins (of the leukocytes). In a majority of the cases it contains also at least a small amount of albumin and perhaps a few casts.

The conspicuous feature of the disease is the great increase in the number of circulating leukocytes. They usually vary from 100,000 to 500,000 when the patient first seeks advice; commonly there is a progressive, though not steady, increase in their number, so that counts of 1,000,000 or more are not rare. This increase is due to an absolute increase of all the normal circulating leukocytes (including lymphocytes) and to a large number of myelocytes. The myelocytes and the polynuclear neutrophiles each vary, as a rule, between 30 and 60 per cent. of the total leukocytes, the polynuclear neutrophiles being usually in excess of the myelocytes; the eosinophiles and the basophiles (mast cells) also show a percentage as well as an absolute increase; the lymphocytes and the so-called large mononuclear leukocytes (variously named), as a rule, show no percentage increase (usually a percentage decrease), but a great absolute increase. All of the leukocytes (including the myelocytes) exhibit various deviations from the normal in size, shape, character of the nucleus, and staining affinities of the protoplasm and of the granules.

The red cells may not be much reduced until the disease has made considerable progress, when marked oligocythemia may occur—2,000,000 or less; so that in advanced cases the ratio of leukocytes to erythrocytes may be 1 to 2, even 1 to 1. The color index is usually low (0.8 or less), as in any secondary anemia. Normoblasts and megaloblasts are always present; they are often polychromatophilic, and may exhibit mitosis. The blood platelets are increased. Charcot-Leyden crystals often separate out from the clotted blood.

Lymphocytic or Lymphoid Leukemia.—Lymphocytic or lymphoid leukemia (lymphemia) is relatively uncommon, and occurs mostly in males (two-thirds of the cases). The acute cases are most common before the thirtieth year, many occurring before the twentieth year; the chronic cases are most common after the thirtieth, especially after the fiftieth year (80 per cent. of the cases).

Pathology.—The lesions consist of lymphocytic or lymphoid hyperplasia of the leukoblastic tissues—the lymph nodes, the spleen, and the bone marrow. The changes likely may begin anywhere in the leukoblastic tissues, but the bone marrow apparently is always involved; in some cases the lymph nodes show no changes whatever. The naked eye appearances are not notably different from those seen in myelocytic leukemia; but microscopically the cellular infiltrations and nodule formations are of the lymphoid type (large or small cell, depending largely upon the acuteness or chronicity of the process).

Symptoms.—The onset may be abrupt or insidious. The acute cases exhibit the common phenomena of an acute infection, with fever (104° to 107°), and its common concomitants, weakness, progressive emaciation, etc. Often the cases are mistaken for typhoid fever, puerperal infection, other types of septicopyemia, etc. Usually there is moderate enlarge-

ment of the palpable lymph nodes, but this may be totally wanting, or it may supervene late in the course of the disease. In some cases the early complaint is of enlarged tonsils, which is usually misinterpreted as tonsillitis (confirmation of this supposition being sometimes found in enlargement and tenderness of the adjacent cervical lymph nodes, the fever, and leukocytosis—without a differential count). Hemorrhages are quite characteristic, and with the other phenomena suggest the disease even without a blood examination. The hemorrhages occur into the skin (petechiæ, ecchymoses), in the mucous membranes, especially the mouth, gastro-intestinal tract, genito-urinary tract, etc.; in the serous membranes, the retina, the viscera, etc.; often, especially in the mouth and the gastro-intestinal tract they are attended by much ulceration and sloughing. The chronic cases usually begin with increasing weakness and enlargement of the lymph nodes—the cervical, the axillary, the inguinal, etc. In some cases there is at first marked enlargement which has developed rapidly, but this gradually subsides and the disorder then goes on chronically; but in some cases, in the absence of glandular enlargement, the diagnosis is first disclosed by an examination of the blood. Hemorrhages are uncommon in the chronic cases. Attacks of fever may occur from time to time.

Examination discloses much the same abnormal physical conditions as are encountered in myelocytic leukemia. The palpable lymph nodes, as a rule, are more or less, sometimes enormously, enlarged (in contrast to the usual slight enlargement in myelocytic leukemia); the mesenteric and retroperitoneal nodes may be palpable; the spleen and the liver, however, are rarely as enlarged as in myelocytic leukemia. The skin, in addition to hemorrhages, may disclose urticarial swellings, lymphomatous nodules, etc.

The diagnostic feature of the disease is the great increase of circuculating lymphocytes. The total leukocyte count varies from normal to 500,000 or more; usually at the first count it varies between 20,000 and 60,000; the average is less than in myeloid leukemia (about 150,000 as compared to about 400,000), but rarely in very acute cases the count may exceed that of the myelocytic variety. The increase consists exclusively of lymphocytes, which may constitute 99 per cent. or more of the total count. The acute cases usually reveal a very high total leukocytic count, high percentage of lymphocytes (90 to 99 per cent.), and preponderance of large lymphocytes; the chronic cases, less high total count, less percentage of lymphocytes (50 to 80 per cent. or more), and preponderance of small lymphocytes. The small lymphocytes doubtless are derived in large measure from the lymph nodes; but the large lymphocytes of leukemia seem to be unquestionably bone-marrow cells, since they differ materially from the normal large lymphocytes of the circulating blood, and much resemble, if they are not identical with, cells variously described as Grawitz's unripe or immature leukocytes, indifferent lymphoid cells (Pappenheim and Wolff), Naegeli's myeloblasts, Türk's stimulation cells, etc.; perhaps they are in reality mother cells,

and to be interpreted as manifestations of reversion to an ancestral type. Some of the small lymphocytes also are distinctly of this type, and often there is no sharp line of distinction between the so-called large and the small lymphocytes. The lymphocytes occasionally exhibit mitotic figures; commonly they show divers retrograde changes. The granular leukocytes are relatively and absolutely reduced; one or the other type may be entirely absent from time to time. Occasionally a few myelocytes may be seen (stimulation myelocytosis). The erythrocytes usually are reduced to between 3,000,000 and 4,000,000 when the patient is first seen; the color index is low, and there are the other evidences of secondary anemia—which increase with the progress of the disease. Erythroblasts are usually present, especially in the acute cases.

Atypical Leukemia.—The term atypical leukemia finds some, but incomplete, justification in certain deviations from the common types exhibited by the two varieties of leukemia and in the occurrence of certain disorders more or less resembling leukemia, or out of which leukemia may develop. In otherwise typical leukemia, the blood picture is sometimes atypical; thus, the typical picture of lymphocytic leukemia may be present with a total leukocyte count of 7500 or less; in myelocytic leukemia the eosinophiles or mast cells may be absent or unusually numerous, and the percentage of polynuclear neutrophiles may be very high or very low; and under the influence of infections, such as influenza, miliary tuberculosis, etc., the leukemic blood picture may become remarkably altered, or it may disappear entirely temporarily. The blood picture often becomes atypical shortly before death. In some cases leukemic or leukemic-like changes occur in the hemopoietic tissues without leukemic changes appearing in the circulating blood (discussed under tumors and tumor-like formations of the lymph nodes); in some of these cases true leukemia with the characteristic blood picture may subsequently develop. Rarely leukemic changes have been found in the blood without leukemic changes in the hemopoietic tissues (doubtless due to imperfect examination). The supposed association of some features of leukemia with some features of pernicious anemia (leukanemia, Leube) has already been mentioned. Only rarely, and then doubtfully, do any of these conditions justify the term atypical leukemia.

Chloroma.—Chloroma is an atypical form of lymphocytic leukemia in which the leukoblastic hyperplasias take on a greenish tint and in which some of those growing in the bone marrow penetrate the bone and give rise to veritable tumor formations that invade the surrounding tissues. The tumor-like growths are found most commonly in the skull, orbit, ribs, sternum, vertebræ, etc., but also in the long bones and in the lymph nodes, the viscera (kidneys, liver, spleen), the skin, etc. They give rise to local pain (headache, pains in the legs, etc.), regional swellings (especially in the temporal region, the orbit, etc.), deafness, exophthalmos, blindness, etc. The general manifestation, such as weakness, emaciation, hemorrhages, anemia, enlargement of the lymph nodes, the spleen, and the liver, and the blood picture are quite like those of lympho-

cytic leukemia; rarely they have been said to resemble myelocytic leukemia. The disorder is usually fatal within four or six months; occasionally it has been protracted to a year or more.

Diagnosis.—The diagnosis of leukemia is often suggested by enlargement of the spleen or the lymph nodes (especially significant if associated with hemorrhages), but certainty is obtained only by an examination of the blood. One should distinguish between acute and chronic cases: myelocytic leukemia is almost always chronic; lymphocytic leukemia may be acute (large cell variety) or chronic (small cell variety). Acute cases are also characterized by fever, hemorrhages, and marked fluctuation in the number of circulating leukocytes and in the size of the enlarged lymph nodes and the liver and the spleen. One must exclude typhoid fever, which the acute cases resemble in the fever, typhoid state, enlarged spleen, perhaps intestinal hemorrhages (and the ulceration of Peyer's patches that may be found after death). Different types of septicopyemia (acute endocarditis, puerperal infection, liver abscess, etc.), scurvy, hemorrhagic purpura, etc., also must be excluded; in many cases the diagnosis is not possible without an examination of the blood. The lymphocytosis that sometimes occurs in certain infectious diseases, notably pertussis, but also syphilis, malaria, etc., and the myelocytosis that may occur in infections, such as diphtheria, scarlatina, etc., and in certain forms of secondary anemia (notably von Jaksch's disease), should not be confused with leukemia. Care should be taken also not to overlook the disease in the beginning when the leukocytosis may be very slight, or later, during a period of remission, when there may be a fall to almost, if not quite, the normal number of leukocytes; a differential count will furnish the clue to the correct diagnosis. The differentiation from Hodgkin's disease and other disorders attended with enlarged lymph nodes is discussed under tumors and tumor-like formation of the lymph nodes.

Prognosis.—The disease is always fatal—within a week to two or three months in the acute cases; within from six months to three or more years in the chronic cases. In the acute cases death usually ensues in consequence of fever, hemorrhages, anemia, and exhaustion. In the chronic cases, remissions for weeks or months are not uncommon (spontaneous or due to intercurrent infection, treatment, etc.). Death may result from the ordinary progress of the disease, which toward the end is likely to be rapid, or from an intercurrent infection, such as tuberculosis, pyococcic infection, influenza, pneumonia, etc.

Treatment.—The nature of the disease, so far as we know, seems to preclude effective treatment. The acute cases pursue, as a rule, a steadily progressive course to the fatal issue, although rarely a case beginning apparently acutely becomes stayed and goes into the chronic stage. In the chronic cases arsenic and the *x*-rays are often of much benefit—arsenic in both varieties of the disease, the *x*-rays especially in the myelocytic variety. The arsenic should be given in gradually ascending doses to the point of toleration; sometimes benefit is not apparent until the

dose reaches a dram of Fowler's solution daily. The *x*-ray treatment should be carried out as recommended by Stengel and Pancoast systematically: the bone marrow in different regions in succession should be subjected to the action of the *x*-rays for periods averaging fifteen minutes daily; the spleen should not be subjected to the direct action of the *x*-rays until it has become reduced in size and the total leukocyte count has diminished; special care must be exercised to avoid even the slightest toxic reaction. The treatment should be continued until the spleen and the leukocyte count are normal; discontinuance of the treatment should be gradual, and the period of cessation should not be too protracted. The *x*-rays exert a specific leukolytic action, especially on the young cells—the non-granular mononuclear leukocytes and the myelocytes; and the serum of a leukemic subject that has improved under *x*-ray treatment contains a substance which injected into lower animals produces leukopenia, disintegrating, first, the mononuclear cells; perhaps the future will furnish us a leukolytic serum efficient in human subjects. Splenectomy in leukemia, of course, is an irrational measure.

ERYTHREMIA.

(*Erythrocytosis; Megalosplenic Polycythemia or Erythrocythemia; Polycythemia with Cyanosis and Enlarged Spleen; Vaquez's Disease.*)

Erythremia is a disease of the hemopoietic tissues, characterized by hyperactivity of the erythroblastic tissues of the bone marrow, and by polycythemia, chronic cyanosis, and enlarged spleen.

Etiology.—The disease occurs with about equal frequency in the two sexes, and especially after the thirty-fifth year. The cause is not known. Some of the early cases were attributed to tuberculosis of the spleen, but doubt has been thrown upon the accuracy of the observation. Hyperactivity of the erythroblastic tissues of the bone is believed to be the basis of the disorder, but the initiating cause is unknown.

Pathology.—The noteworthy changes consist of a purplish-red bone marrow (myelomatosis rubra) due to extraordinary hyperplasia of the erythroblastic tissues; and enlargement of the spleen due to hyperplasia of all the constituent elements, and some evidences of chronic passive congestion. The changes in the spleen are believed by some observers (Osler, etc.) to be active rather than passive; perhaps a reversion to the fœtal condition of erythrocytogenesis; others (Widal, Weintraud, etc.) suggest that the spleen loses its function of hemolysis, in consequence of which erythrocytes accumulate and the erythroblastic tissues of the bone marrow become abnormally stimulated. The disease has been attributed also to diminished oxygen absorption by the erythrocytes and consequent hyperplasia of the erythroblastic tissues.

Symptoms.—The symptoms begin gradually, as a rule, in middle life or later; they consist of gradually increasing weakness, headache, vertigo, hebetude, and other symptoms attributable to cerebral congestion;

chronic cyanosis; and enlargement and fulness of the abdomen, with a sense of weight, especially in the region of the spleen. The skin in general, but especially of the face, hands, and feet, and the visible mucous membranes take on a peculiar, mottled, purplish-red color; the lips, ears, and finger tips become even darker, more dusky. The color, especially of the limbs, becomes darker under the influence of cold (varying thus with the seasons) and of gravity; and it lessens if the arms or legs be held upright (vasomotor instability). The cyanosis is attributable to the polycythemia and increased volume of blood, increased viscosity of the blood, slow circulation, and distended venules and capillaries (loss of tonicity). In long-standing cases pigmentation of the skin and mucous membranes ensues. Hemorrhages from the mucous membranes (nose, mouth, lungs, stomach, etc.) are not uncommon (stasis and impaired nutrition); occasionally they occur in the viscera, notably the brain, where they give rise to the ordinary phenomena and sequels of apoplexy. The fulness in the abdomen is due to an enlarged spleen, which is present in at least four-fifths of the cases. The liver also may be somewhat enlarged. The heart may become hypertrophied. The blood pressure is usually normal. Examination of the blood reveals increased (in some cases doubled) total volume, increased specific gravity (up to 1077), and increased viscosity (3 to 4 times the normal); the erythrocytes are increased up to 9,000,000 to 13,000,000 per cubic millimeter, the hemoglobin to 120, 150, even 200 per cent., and the leukocytes to 10,000 or 20,000 or more. In a majority of the cases there is a relative increase of the polynuclear neutrophiles (75 to 90 per cent.), and sometimes of the large mononuclear leukocytes; occasionally myelocytes are seen; the erythrocytes, as a rule, are normal in size, but there are some poikilocytes and many erythroblasts (normoblasts and megaloblasts). The blood changes together indicate hyperactivity of the bone marrow.

Diagnosis.—Chronic cyanosis, enlarged spleen, and erythrocythemia constitute a triad of symptoms that is almost pathognomonic. One must eliminate other causes of chronic cyanosis, such as chronic cardiac and pulmonary disease, the methemoglobinemia of poisoning by coal tar products, so-called enterogenous cyanosis, local asphyxias (Raynaud's disease and allied conditions), etc.

Prognosis.—So far as is known the disorder is ultimately fatal. It usually pursues a chronic course, upward of six to eight years or more.

Treatment.—No really effective treatment is known. Removal of the spleen seems contra-indicated from the nature of the disease and the tendency to hemorrhage. Venesection is of value in relieving temporarily the headache, vertigo, etc.; it may be repeated from time to time. Potassium iodide has been advocated, and oxygen by inhalation has been used with a view to relieve the cyanosis, but both are of at least doubtful utility. The *x*-rays, as in leukemia, constitute perhaps the best treatment, but the data at hand are not yet sufficient to warrant a positive opinion.

THE HEMORRHAGIC DISEASES.

HEMORRHAGE IN THE NEWBORN.

Hemorrhage occasionally occurs in the newborn, and usually from the umbilicus, the gastro-intestinal tract, the rectum, the mouth, the nose, the bladder, etc. It is commonly associated with jaundice. It may be due to: (1) Injuries received during birth. (2) Septicopyemia acquired during or after birth. (3) Syphilis (syphilitic arteriocapillary disease), in which event it may be present even at birth, and may be associated with visceral hemorrhages. (4) Epidemic hemoglobinuria (Winckel's disease), a disorder of unknown, though probably infectious, nature (hospital epidemics), developing usually within the first four to ten days of life, and characterized by fever, jaundice, punctiform hemorrhages in the tissues and organs, dyspnœa, cyanosis, gastro-intestinal derangements (diarrhœa), enlarged spleen, albuminuria, hematuria or methemoglobinuria, etc., and soon leading to death. In some cases there is marked fatty degeneration of the viscera (Buhl's disease). (5) Morbus maculosus neonatorum, a disorder clinically akin to morbus maculosus of the adult, and as ill understood etiologically, although it is perhaps infectious (sometimes attended by fever, occurs especially in hospitals, and sometimes appears to be self-limited). The bleeding is perhaps most common from the bowel (melena—which should not be confounded with blood sucked from the breast); it may also occur from the stomach, mouth, nose, umbilicus, etc. Death occurs in 50 per cent. or more of the cases. The *treatment* of these disorders is that of syphilis or septicopyemia, to which may be added the subcutaneous injection of normal human or other blood serum and such local measures (mentioned under hemophilia) as may tend to control the bleeding.

PURPURA.

The term purpura comprises a series of disorders characterized by the occurrence of hemorrhages into the skin and mucous membranes. Commonly, like anemia, it is symptomatic of some more or less obvious disorder, but often it partakes of the nature of an independent disease of unknown cause. One may distinguish, therefore, two types—the secondary or symptomatic, and the primary or idiopathic.

The hemorrhages, so-called purpuric spots, vary in size—from small roundish petechiæ, 1 to 3 mm. or more in diameter, to larger ecchymoses, often irregular in outline and perhaps due to confluence of several small areas. Occasionally the hemorrhages are linear (vibices). When fresh they are bright red in color, and being extravasations they do not disappear on pressure; soon they become dark or purplish, and gradually transformed into reddish-brown or yellowish areas, so that at the end of a week, as a rule, only more or less faintly pigmented spots remain.

Successive crops occasion considerable color variation of adjacent spots. Most frequently the legs are involved, and then in order of frequency the arms, the trunk, the face, and the accessible mucous membranes.

Secondary (symptomatic) purpura occurs in: (1) Infections, such as typhus fever, cerebrospinal fever, and acute endocarditis—all three of which are especially characterized by a purpuric eruption; it may occur also in septicopyemia (pyococcic infections), smallpox, typhoid fever, scarlatina, measles, syphilis, cholera, etc. (2) Intoxications, such as snake bite; drug poisonings, especially with the iodides, but also bromides, quinine, salicylic acid, belladonna, copaiba, ergot, mercury, arsenic, etc.; and autogenic poisonings, such as may attend nephritis, jaundice, etc. (3) Cachectic states or nutritional disorders, such as are associated with the marasmus of old age (senile purpura), general inanition, carcinoma, sarcoma, pernicious anemia, leukemia, Hodgkin's disease, etc. (4) Nervous disorders (myelopathic purpura), such as myelitis, tabes dorsalis (in which it may follow and be confined to the area of the lightning pains), hysteria (hemorrhagic stigmas), neuralgias, such as trifacial neuralgia and sciatica (in which the purpura may be limited to the painful area), etc. (5) Mechanical disorders, such as marked venous stasis (as in heart disease, following the application of a tight bandage, etc.), embolism, epileptic seizures, whooping-cough paroxysms, local injuries, etc. Mechanical factors seem operative in other types of the disorder, and perhaps explain the frequency of the petechiæ on the legs.

Primary (idiopathic) purpura comprises several disorders, variously described as simple purpura, arthritic (rheumatic) purpura, hemorrhagic purpura, abdominal (Henoch's) purpura, etc. These differ perhaps in grade, intensity, or localization of the process rather than in nature. In the present state of our knowledge it is impossible sharply to differentiate them or satisfactorily to classify them.

Etiology.—The disorder is most common in the second and third decades of life, although it occurs at all ages, and it affects males rather than females. The cause is not known. By some it is believed to be an infection or an intoxication, but neither the infectious agent nor the toxin has been isolated; blood cultures have usually been negative. Primary disturbances of the nervous system, of the liver, of the blood, and of the bloodvessels have been postulated. Judging from the perhaps analogous action of the hemorrhagin of snake venom, it seems likely that the essence of the process consists in an endotheliolysis of the capillaries provoked by some unknown poison; the primary change may occur in the blood.

Pathology.—There are no characteristic lesions, aside from the hemorrhages. In fatal cases there is high-grade anemia of the viscera with the usual concomitants. There may also be petechial and larger hemorrhages into the serous and mucous membranes. The spleen may be enlarged, and there may be nephritis (usually glomerulo-nephritis). No constant or characteristic changes have been found in the bloodvessels. During life the blood reveals the ordinary changes of secondary

anemia, although in some cases the erythrocytes become reduced much below 2,000,000 (to about 500,000 in some fatal cases); the blood platelets are usually remarkably reduced (20,000 or less instead of the normal 200,000 to 700,000). The coagulation time of the blood is usually within normal limits, although it may be considerably increased (delayed); the clot often is less firm than normally.

Symptoms.—In the simplest form (so-called purpura simplex), which is most common in adult life, the cutaneous hemorrhages, as a rule, are the only noteworthy manifestation. They are usually small (petechial), occur in crops, and are most common on the legs and arms. Occasionally there is a little fever or slight pain and tenderness in the joints and muscles. The disorder usually lasts from two to six weeks; rarely there are recurring attacks that last for a year or more (chronic purpura).

When the articular symptoms are obtrusive the disease is usually spoken of as purpura (or peliosus) rheumatica (or arthritica), or Schönlein's disease, but it is in no way related to acute articular rheumatism. In about half of the cases the onset of the disease is attended with fever (101° to 102°), and there may be prodromal or invasion symptoms consisting of sore throat (tonsillitis), malaise, anorexia, nausea, neuromuscular and articular pains and tenderness, etc. The purpuric eruption is like that of simple purpura, perhaps somewhat aggravated. It may be preceded by or associated with urticaria (purpura urticans), or erythema nodosum, or rarely vesiculation (pemphigoid purpura). The articular symptoms consist of pain, tenderness, and swelling; they are most common in the knees, ankles, and elbows, but may occur in other joints; they may antedate or occur with or subsequent to the purpura; and they usually last a few (four to seven) days, but may recur.

The curious association of purpura, urticaria, erythema, angioneurotic œdema, and visceral lesions has been especially studied by Osler, who states that it occurs chiefly in children, and is characterized by relapses or recurrences, often extending over several years; cutaneous lesions, which may be simple purpura, purpura urticans, urticaria, angioneurotic œdema, and erythema in all its multiform varieties; gastro-intestinal crises—pain, vomiting, and diarrhœa; joint pains or swelling, often trifling; hemorrhages from the mucous membranes; enlargement of the spleen; and nephritis (40 to 50 per cent. of the cases), which is the most frequent cause of death (which occurs in about 7 per cent. of the cases). The cases characterized by purpura or other skin lesions, gastro-intestinal crises, arthritis, and often nephritis, are commonly spoken of as Henoch's or abdominal purpura.

In another series of cases, to the cutaneous and arthritic lesions hemorrhages from the mucous membranes are added (hemorrhagic purpura, morbus maculosus, Werlhof). Nasal and oral hermorrhages are the most common; but there may be bleeding from the lungs, stomach, intestine, kidney, genitalia, or rarely into the retinæ, brain, etc. Occasionally cases have been observed in which, for a time at least, the cutaneous manifestations were absent, while the other symptoms (such as

visceral crises, epistaxis, etc.) seemed to justify the diagnosis (so-called purpura without skin lesions); in some of these cases skin lesions have subsequently appeared. The disease may terminate favorably within two weeks or thereabouts; but death may ensue from the direct loss of blood, hemorrhage into the brain, etc. Occasionally in very severe cases death occurs within twenty-four or thirty-six hours or thereabouts (purpura fulminans).

Diagnosis.—One should distinguish the symptomatic from the so-called idiopathic cases. The idiopathic cases may be distinguished from hemophilia by the absence of hereditary factors; and from scurvy by a knowledge of the previous history, the absence of sponginess of the gums (bleeding from the gums is not uncommon in purpura), the absence of the hemorrhagic, sclerodermic, or muscular infiltration of the thighs (so characteristic of scurvy), and the effect of antiscorbutic treatment.

Prognosis.—The outlook is good except in the event of severe onset and rapid course (purpura fulminans), very great loss of blood, hemorrhage into the brain, nephritis, and other serious complications.

Treatment.—The treatment of symptomatic purpura is that of the primary or underlying disease. In the idiopathic cases rest in bed, fresh air, good food, iron, bitter tonics, strychnine, etc., are indicated. Good results sometimes attend the use of arsenic in gradually increasing doses to the point of toleration; and of aromatic sulphuric acid and turpentine (which seem to check the bleeding), and of calcium lactate (to increase the coagulability of the blood). Accessible bleeding spots may be treated as recommended in hemophilia.

HEMOPHILIA.

Hemophilia is a disorder, commonly hereditary, characterized by severe and often uncontrollable hemorrhage and lessened coagulability of the blood, and often associated with hemarthrosis and arthritis.

Etiology.—Hemophilia is "the most hereditary of hereditary diseases" (Grandidier), and, as in the Appleton-Swain family, is known to have been transmitted to the seventh generation (within a period of two hundred years). Males usually exhibit the disorder—the proportion of males to females varying between 13 to 1 (Grandidier, 657 cases) and 4 to 1 (Stempel, 209 cases); but the disease seems to be transmitted almost exclusively by the females. The Anglo-German races are most frequently attacked, but even negroes are not exempt. Hemophilic subjects usually are otherwise healthy; commonly they are blondes with soft delicate skin; often they are neurotic; usually they are remarkably fecund.

The cause of the disease is not known. There is no good evidence that consanguinity is of etiological significance. Virchow believed in an abnormal tenuity and fragility of the vessels, but no noteworthy or constant changes are found. Sahli suggests an hereditary deficiency in the

formation of thrombokinase by the vessel wall. An infectious theory has been advanced, but it is not supported by any trustworthy evidence.

Pathology.—The only constant finding in fatal cases is high-grade anemia and its usual consequences. In a few cases the arteries have been found unusually thin; in others the endothelium of the arterioles has proliferated; in still others the left ventricle has been hypertrophied—but none of these changes appears to possess any special significance.

Symptoms.—In about three-fourths of the cases the disorder becomes manifest in the first two years of life (rarely in the newly born); occasionally it is delayed until the second or the third decade, rarely until advanced life. The characteristic of the disease is severe, often uncontrollable hemorrhages, occurring spontaneously or induced by slight (or pronounced) trauma. The hemorrhages may be superficial—into or from the skin and the mucous membranes; interstitial or parenchymatous—into the subcutaneous and submucous tissues and the organs of the body; or they may occur into the serous sacs. The chief sources of the bleeding are the nose (50 per cent. of the cases), the mouth and gums (12 per cent.), the stomach, intestines, lungs, kidneys, skin (where in contrast to purpura they are rarely petechial), etc.; but they may occur from any part of the body, such as the urethra, vulva, uterus, tear papilla, eyelids, etc. The hemorrhage is usually a capillary oozing only, but it may be also arterial or venous, especially in the event of incised or lacerated wounds. The bleeding may last for hours or days, and on account of the large amount of blood lost may promptly lead to death; rarely the bleeding continues for months. The retained hemorrhages may give rise to smaller or larger hematomas. Minor operations, such as circumcision, the eruption or extraction of a tooth, vaccination, etc., may give rise to severe and even fatal hemorrhage; the hemorrhage of menstruation and of parturition may be excessive, but is usually not unless there are other phenomena of the disease. Joint manifestations, involving especially the knees and elbows, but also other joints, are exceedingly common; according to König they may be: (1) Hemarthrosis (pain, tenderness, swelling, perhaps fluctuation, and visible hemorrhage about the joint); (2) arthritis, with fever, resembling tuberculosis; and (3) a condition resembling arthritis deformans. There may be associated muscular pains, neuralgias, etc. In some cases the tendency to bleeding seems to be confined to some particular part of the body (local hemophilia), such as the kidney (so-called renal hemophilia), the gastro-intestinal tract, the nose, etc., other parts of the body apparently being (temporarily or permanently) exempt. Some of the cases of recurring epistaxis are associated with curious multiple hereditary telangiectases of the nose, lips, tongues, cheek, etc.

Following the bleedings there are the usual changes of secondary anemia. There may be a relative lymphocytosis. The coagulability of the blood is usually lessened; in some cases it has been found remarkably lessened (the coagulation time being forty-five to sixty minutes or more); but recent studies by Sahli tend to show that the coagulability

may be normal or increased during the period of bleeding and lessened during the intervals. If the hemorrhage does not prove fatal, recovery is usually more rapid than after other types of bleeding.

Diagnosis.—The familial transmission of the disease, its common development in early life, its usual persistence throughout life, the occurrence and recurrence of bleeding upon little or no provocation, the disproportion of the bleeding to the provoking trauma, the virtual uncontrollability of the bleeding, and the joint phenomena are quite characteristic, and serve to exclude the hemorrhages of the newborn, purpura, and scurvy.

Prognosis.—Any hemorrhage may be, but the first is rarely, fatal. Many if not most subjects die within the first year of life, and more than 80 per cent. before reaching adult life. The outlook is worse in boys than in girls.

Treatment.—With a view to preventing the disease, the daughters of hemophiliacs should be advised not to marry—advice that is likely not to be heeded. The boys should avoid traumas of all sorts; they should be rendered as robust as possible by fresh air, good food, hydrotherapy, massage, tonics, etc., and operations, even minor ones, such as circumcision, extraction of a tooth, etc., should be interdicted, except in the event of diseases otherwise threatening life.

When hemorrhage has occurred, it may sometimes be controlled by rest and compression, such as plugging the nares, or the application of compresses to other accessible regions. Adrenalin chloride (1 to 1000), ice, cocaine (2 to 4 per cent.), calcium lactate or chloride (2 per cent.), gelatin (5 per cent. effectually sterilized), gallic acid, tannic acid, and other so-called styptics may be applied locally (on the compress); but usually they are of little avail. Wright recommends the inhalation of carbon dioxide and oxygen in epistaxis. Internally calcium lactate (15 to 20 grains, 1 to 1.3 gram, thrice daily, or 50 grains, 3.3 gram, at once) may be given. It is wise to intermit its administration after a few days, since continuous use sometimes seems to lessen, after having first increased, the coagulability of the blood. The best results appear to attend the use of normal blood serum (of the rabbit, horse, or man) locally, subcutaneously, and intravenously. A single injection subcutaneously of 20 to 30 c.c. has sufficed to arrest the bleeding, but it may be necessary to repeat the injections daily or every two or three days until the desired result has been secured. In the absence of other serum, the ordinary antidiphtheritic serum may be used. The arthritic lesions should be treated by rest (immobilization), and after several days by massage. Much swelling and pain may necessitate aspiration. Extreme loss of blood demands measures appropriate for exsanguination occurring under other circumstances, such as physiological saline solution intravenously, adrenalin chloride, etc. (see acute secondary anemia).

DISEASES OF THE LYMPH NODES AND LYMPH VESSELS.

Pathological Physiology.—The lymph nodes, as has been mentioned, are concerned in hemopoiesis to the extent of forming perhaps most of the circulating lymphocytes during health; to some degree also they serve to protect the body against bacterial infections; they have slight phagocytic properties; and they participate in the processes of hemolysis. The formation of lymphocytes becomes extraordinarily augmented in some cases of leukemia, in which event the nodes exhibit various degrees of hyperplasia and metaplasia. The protection exerted against bacterial infection is not, as a rule, highly effective, and may become totally overcome, especially in severe pyococcic infections. In other cases, however, doubtless in consequence of chemical activities, but perhaps also of phagocytic properties, bacterial activity is restrained, if not entirely abolished; but, as is well exemplified in some cases of tuberculosis, bacteria may remain for a long time latent in lymph nodes, and under auspicious circumstances become activated. The hemolymph nodes, certain nodes especially numerous in the abdomen in front of the vertebræ, and containing, in addition to the structures of the ordinary lymph nodes, sinuses filled with blood corpuscles, in general subserve the same functions and are subject to the same pathological conditions as the lymph nodes. In certain disorders, such as pernicious anemia and some toxic and infectious processes, they seem to be unduly concerned in hemolysis as well as in processes of immunity (Warthin). Whether these glands participate also in erythrocytogenesis has not yet been determined. The lymph nodes may entangle metastatic carcinomatous cells, and enlarging may be the first noteworthy evidence of internal carcinoma.

LYMPHANGITIS AND LYMPHADENITIS.

Lymphangitis and lymphadenitis, although they may occur separately, are usually associated; the process may be acute or chronic, and is always due to bacterial infection. The acute cases are usually due to the ordinary pyogenic cocci—staphylococci, streptococci, pneumococci, etc.; but acute lymphadenitis is common also in many of the acute specific infectious diseases, such as diphtheria, scarlatina, measles, chickenpox, erysipelas, plague, glandular fever, typhoid fever, gonorrhœa, chancroid, as well as rickets, lymphatic constitution, etc.; the chronic cases are due usually to tuberculosis or syphilis. The infection in lymphangitis is usually acquired by means of a wound, but this is not always necessary, since infection seems sometimes to be acquired without or with only the slightest abrasion, as at necropsies, operations on septicopyemic patients, etc. The infectious agent may enter the lymph vessel

directly from the wound, or it may invade the vessel wall from without. In lymphadenitis the infection may be lymphogenous, in which event it is likely to be localized; or it may be hematogenous, in which event it is likely to be generalized (as in some of the specific infectious diseases).

Pathology.—Acute lymphangitis is commonly a part of pyococcic infection (septicopyemia) and partakes of the nature of such infections. It may involve only circumscribed areas (reticular lymphangitis), or the lymphatic trunks (tubular lymphangitis). The endothelial cells lining the lymph spaces swell and later desquamate, and together with leukocytes, serum, and fibrin obstruct the vessel. This inflammatory exudate commonly coagulates and the vessel becomes completely thrombosed and surrounded by more or less inflammatory congestion and œdema (perilymphangitis); occasionally small foci of hemorrhage supervene. Resolution, suppuration, or organization may ensue, depending upon the virulence of the infecting agent and the resistance of the individual. In the event of suppuration, phlegmonous infiltration may follow. In the event of organization, proliferation and thickening of the vessel wall and complete obstruction of the lumen usually supervene.

In most cases the lymph nodes participate in the process and local or general lymphadenitis results. The local process is well seen in infections of the hands and arms (axillary nodes) or of the feet and legs (femoral nodes); in infections of the mouth, carious teeth, facial erysipelas, tonsillitis, etc. (cervical nodes); in pneumonia, bronchitis, especially in children, tuberculosis, etc. (bronchial nodes); in appendicitis, typhoid fever (mesenteric and retroperitoneal nodes); in gonorrhœa and chancroid (inguinal nodes, so-called bubo); etc. The general process is common in the aforementioned infectious diseases, but occurs notably in glandular fever, tuberculosis, syphilis, plague, etc. In the acute cases the nodes become enlarged, swollen, soft, and hyperemic; resolution may occur, or liquefaction, necrosis, and suppuration may ensue. Microscopically there is lymphoid hyperplasia, vascular dilatation, polynuclear leukocytic infiltration, fibrin formation, and swelling and desquamation of the endothelium of the sinuses. Considerable necroses may occur, as in typhoid fever; or the process may become hemorrhagic or purulent. In the chronic cases there is a low-grade, chronic, inflammatory process, with the formation of cicatricial connective tissue, in consequence of which the nodes become firm, hard, and small; the capsule becomes thickened, the sinuses more or less obliterated or obstructed, and the lymphoid tissue atrophic.

Symptoms.—Reticular lymphangitis manifests itself by circumscribed, red, swollen, painful, and tender areas, which are commonly seen on the arms or legs, and progress usually from the periphery toward the centre; frequently there is considerable collateral œdema. Tubular lymphangitis manifests itself in cord-like, irregular, reddish streaks beneath the skin, progressing from the periphery toward the regional glands. As already stated, both varieties are commonly associated, and there is usually concurrent inflammation of the regional lymph nodes—

which become swollen, painful, and tender. The general symptoms are those of pyococcic infection—malaise, chilliness, and fever, in degree depending upon the severity of the infection. In the event of suppuration, the common symptoms of septicopyemia (chills, fever, sweats, and leukocytosis) supervene, and if the lymph nodes are involved they become softened and fluctuating, and may rupture. In the event of chronic proliferation the lymph vessels become thickened, cord-like, and chronic œdema due to obstruction occurs, unless a collateral circulation develops. If a large trunk is involved a considerable area of the body may become chronically œdematous or elephantiatic, or chyluria or lymphorrhagia, etc., may ensue.

Involvement of the deep intrathoracic and intra-abdominal lymph vessels and nodes may be difficult or impossible of diagnosis, since, aside from local pain and perhaps tenderness, symptoms may be absent or in abeyance. The considerable enlargement of the bronchial nodes that sometimes occurs in children is suggested by dyspnœa, respiratory stridor, sometimes dysphagia, and the physical signs of bronchial stenosis (dulness over the vertebrae, *x*-ray examination, etc.). Sudden death occasionally ensues from suppuration and rupture into the trachea or a bronchus.

The general lymphadenitis of the acute specific infections is manifested by swollen, painful, and tender nodes occurring during the course of these infections. In the chronic cases, there may be only enlargement of the lymph nodes; sometimes they are more or less tender, and they may become quite hard.

Diagnosis.—In the acute cases phlebitis usually may be excluded by the fact that in lymphangitis the redness is more marked and more superficial and follows the lymph vessels (rather than a vein), and the deep-seated cord-like induration in phlebitis or a thrombosed vein is absent; in addition the neighboring lymph nodes are usually involved. In the chronic cases one must differentiate between the divers sorts of glandular enlargement (see tumors and tumor-like formations). Sometimes it may be necessary to excise one of the enlarged lymph nodes in order to come to a definite conclusion.

Prognosis.—The prognosis depends entirely upon the nature and severity of the infection and the resistance of the patient.

Treatment.—In the acute cases the treatment is that of the primary pyococcic infection together with rest to the affected part, the local applications mentioned in connection with erysipelas, etc. Surgical measures eventually may be necessary.

LYMPHANGIECTASIS.

(*Elephantiasis.*)

Lymphangiectasis, dilatation of the lymph vessels, may be congenital or acquired. The congenital variety results from mechanical or inflam-

matory obstruction of the lymph vessels occurring during intra-uterine life; but some cases developing in adult life without an obvious cause seem perhaps referable to a congenital or inherited predisposition. The acquired variety may be: (1) Parasitic, due to obstruction of the vessels by Filaria bancrofti—the different forms of tropical and subtropical elephantiasis, chylous ascites, chyluria, etc., the result of obstruction of the thoracic duct, etc.; and (2) non-parasitic, which occasionally results from inflammation and thrombosis of the lymph vessels (erysipelas, eczema, infected leg ulcers, etc.); or it may be secondary to cicatricial occlusion of the vessels, to the pressure of tumors, to the blocking of the lumen by tuberculous or carcinomatous material, to the extirpation of lymph nodes, etc. The condition, however, is rare, despite the frequent operation of the causative factors, since such is the free communication between the lymphatics that a collateral circulation is readily established.

Symptoms.—The dilated vessels, noticeable only when situated superficially, that is, in the skin (and due then to obstruction of the deeper lymphatics), appear as soft, transparent sacs or vesicles, often arranged in clusters, and filled with clear or milky lymph. These, which are sometimes spoken of as circumscribed lymphangiomas, are found most commonly in regions rich in superficial lymph plexuses, especially along the inner aspect of the thigh, about the genitalia, on the anterior abdominal wall, in the mesentery (chyle or lymph cysts), etc. Rupture of the vesicles is not infrequent, and sometimes results in the escape of considerable lymph (lymphorrhagia, chylous ascites, etc.). In some cases the obstruction produces diffuse or widespread dilatation of the lymph vessels—usually of the leg or the external genitalia (elephantiasis, lymph scrotum, lymph vulva), although it may occur anywhere. The limb (or other portion of the body) becomes greatly swollen, hard, indurated, and non-reducible—a condition of branny induration, due to hyperplasia and connective-tissue overgrowth of the lymph vessels, skin, and subcutaneous tissues, and chronic œdema from obstruction of the lymph vessels. The limb may be soft and doughy (elephantiasis mollis), or quite hard (elephantiasis dura); and the overlying skin may be thin, smooth, and translucent, or quite thick and horny (pachydermia). Ulceration may supervene, and permit of the escape of more or less lymph—a turbid, milky-white fluid.

Prognosis.—The outlook is not especially hopeful: the condition is usually permanent.

Treatment.—In the circumscribed form relief may sometimes, but rarely, be obtained by extirpation of the dilated vessels; but when the condition is diffuse, temporary relief only may be afforded by massage, an elastic bandage, and elevation of the limb—in the hope that a sufficient collateral circulation ultimately may be established.

TUMORS AND TUMOR-LIKE FORMATIONS.

The lymph vessels are not infrequently the seat of tumor formations, of which, two types may be distinguished—lymphangioma and endothelioma (lymphangiosarcoma). The lymphangioma, which ordinarily is classed as a benign tumor, consists of multiplied and dilated lymph spaces (telangiectatic or cavernous) lined with endothelium and containing lymph. They are most common in the skin and subcutaneous tissues, but they occur also in the internal organs, the mesentery, etc. Common examples are the small plexiform lymphangiomas of the skin, macroglossia, macrocheilia, and certain cystic tumors of the neck, mesentery, etc. The endothelioma is a tumor developed from the endothelium of lymphatic spaces, and is found most commonly in the serous membranes, the salivary glands, the skin, the ovary, the testicle, etc. In addition, the lymph vessels serve as the common source of the local and distant spread of carcinoma, rarely also of sarcoma.

The lymph nodes also are the seat of a number of tumors and tumor-like growths, which have been variously described as lymphoma, lymphadenoma, lymphocytoma, lymphosarcoma, malignant lymphoma, leukosarcoma, lymphomatosis, adénie, pseudoleukemia, Hodgkin's disease, etc. Not only is there much confusion in regard to the identity of the disease or diseases comprised in these terms, but also in regard to their relation or supposed relation to leukemia, on the one hand, and to syphilis and tuberculosis of the lymph nodes, on the other hand. In the majority of cases, however, one can separate with tolerable ease and certainty the lymphatic enlargements due to syphilis or tuberculosis; and ordinarily the enlargements due to leukemia present no diagnostic difficulties. Most of the other lymphatic enlargements seem to be more or less related clinically and pathologically; studying them generally, they seem to exhibit gradations and stages of transition. Thus, there is undoubtedly a leukemic and an aleukemic lymphoma; that is, a disorder characterized by enlarged lymph nodes with or without the leukemic blood picture; the one disease is leukemia in a true sense, the other may be spoken of as pseudoleukemia or Hodgkin's disease. Differentiation is rendered difficult by the fact that, as pointed out by Reed, Longcope, and Warthin, Hodgkin's disease seems to have a characteristic histological picture; that the other cases, of which the histological characteristic is hyperplasia of lymphoid tissue, sometimes do and sometimes do not exhibit the leukemic blood picture, that is, aleukemic lymphomas sometimes progress to true leukemia (perhaps early and late stages of the one disease); and that true leukemia, during the not unknown periods of regression (occurring spontaneously, or induced by treatment), may exhibit the blood picture of aleukemic lymphoma (sometimes, however, the blood is qualitatively if not quantitatively leukemic). Furthermore, there are transition stages between some of these cases, especially the aleukemic lymphomas, which sometimes are spoken of as benign lymph-

omas, and so-called malignant lymphomas; that is, there is no sharp line of demarcation between the cases of lymphoid hyperplasia that do not and those that do infiltrate and ultimately penetrate the capsules of the lymph nodes and invade the adjacent tissues. The difference between the benign and the malignant lymphoma (lymphosarcoma) seems to be one of degree only. On the other hand, the ordinary varieties of sarcoma (round-cell, spindle-cell, angiosarcoma, etc.) may implicate the lymph nodes; they differ from the lymphomas (lymphoid hyperplasias) in that, as do sarcomas generally, they give rise to hematogenous metastases, and that, as a rule, they do not progressively involve adjacent sets of lymph nodes (as do the lymphomas).

In the present state of our knowledge we may differentiate the following varieties of chronic progressive enlargement of the lymph nodes: (1) Those due to specific infections, such as tuberculosis and syphilis; (2) those due to leukemia, of which there may be a preleukemic, an aleukemic, or a subleukemic stage; (3) tumor formations (lymphomas), among which one may include lymphosarcoma (malignant lymphoma), the ordinary types of sarcoma (round-cell, spindle-cell, angiosarcoma, etc.), as well as secondary or metastatic carcinomatous growths; and (4) Hodgkin's disease. The specific infections and leukemia have already been discussed; some of the cases included under (3) the lymphomas, may be an early stage or a *forme fruste* of, or progress to, leukemia; clinically, it is usually impossible to distinguish between the so-called lymphomas and Hodgkin's disease, except by microscopic examination of an excised lymph node.

HODGKIN'S DISEASE.

Hodgkin's disease is a disorder of the lymph nodes characterized anatomically by a peculiar, chronic, perhaps inflammatory process leading to well-marked fibrosis, and clinically by enlargement of the lymph nodes, progressive anemia, and a fatal termination.

Etiology.—Hodgkin's disease is twice or thrice as common in men as in women, and although it may occur at any age, it is especially common in young adults (fifteenth to thirty-fifth year). The cause of the disease is not known. A number of cases have been attributed to tuberculosis, but there is no doubt that although tuberculosis of the lymph nodes may follow an aberrant clinical and pathological course, the great majority, if not all cases, can be readily distinguished from Hodgkin's disease.

Pathology.—The lesions consist of progressive enlargement of the lymph nodes, which usually involves first the cervical nodes, and then in order of frequency the axillary, the inguinal, the thoracic, and the abdominal nodes. In some cases the disorder seems to begin in the deeper nodes, the superficial nodes being involved later; in other cases the enlargement is for a long time circumscribed to one set of nodes, later

perhaps but sometimes not becoming generalized. The lymph nodes vary much in size; they are smooth and spheroidal in shape, soft, and inelastic (sometimes even fluctuating), or firm and discrete, even the largest masses in many cases being made up of discrete nodules bound together by connective tissue. Occasionally, however, adjacent nodules fuse through penetration of the capsules, and they may become adherent to the overlying subcutaneous tissues and skin. On section the nodules are pinkish and semitranslucent, or grayish or yellowish, especially if softening and fibrosis have occurred. The microscopic appearances, which are characteristic, consist at first of hyperplasia of the lymphoid cells, especially of the germinal centres, with proliferation of the endothelial and reticular cells—a process that much resembles chronic inflammation. The cellular proliferation and thickening of the reticulum soon obscure the normal structures, so that only indefinite remains of the lymphoid tissue can be identified. In addition to lymphocytes, polynuclear leukocytes, plasma cells, and endothelioid cells, eosinophiles and mononuclear and multinuclear giant cells are found in considerable numbers, and are considered quite characteristic (Reed, Longcope, Warthin). Ultimately the fibrosis becomes extreme, so that the nodes become converted almost completely into fibrous tissue. Nodules of similar formation are sometimes found in the spleen (which may become considerably enlarged), in the liver, gastro-intestinal tract, kidneys, lungs, bronchi, thymus, bone marrow, serous surfaces, skin, etc.; these are most likely not true metastases, but (as in leukemia) hyperplasias of previously existing areas of lymphoid tissues.

Symptoms.—The first symptoms, as a rule, consist of painless enlargement of the lymph nodes, which, as a rule, begins in one side of the neck, although it may begin elsewhere; in most cases the cervical, axillary, inguinal, thoracic, and abdominal nodes become progressively involved, in some cases soon, in some cases later; rarely the disease remains localized to the set of nodes first involved. The local mass of enlarged nodes often becomes very large, but, as a rule, the individual nodes can be distinctly palpated, although rarely they fuse and perhaps form adhesions with the superjacent subcutaneous tissues and the skin. The nodes may be soft (sometimes fluctuating) or firm; sometimes, in advanced cases, quite hard. Divers symptoms result from local pressure: œdema and cyanosis of the neck, face, arm, or leg; effusions within the serous cavities; dyspnœa, generalized cyanosis, dysphagia, and other manifestations of mediastinal tumor; jaundice, pyloric obstruction, and other gastro-intestinal disturbances; sciatica and other local pains, etc. In some cases the deep (thoracic and abdominal) nodes are first involved, so that these usually late symptoms of pressure may be the earliest manifestations. The spleen is commonly enlarged, sometimes notably enlarged; the liver also is enlarged in about one-half the cases. The skin may become pigmented (independently of the use of arsenic); pruritus and furuncles are often annoying; lymphomatous nodules

sometimes occur in the skin, and subcutaneous hemorrhages occasionally have been observed.

In the ordinary, slowly progressing cases, the general health, aside from more or less malaise, usually remains for a long time unimpaired. In some cases there is more or less fever: it may be a mild continued fever (as high as 100° or 101°); or especially toward the end it may be high (104° to 105°) with daily remission of three or four degrees and accompanied with chills and sweats; or it may be of the relapsing type (Epstein-Pel)—periods of fever of ten to fourteen days' duration alternating with periods of apyrexia. Secondary anemia, ultimately of high grade (2,000,000 erythrocytes or less, 30 to 40 per cent. of hemoglobin), emaciation, and cachexia gradually develop—sometimes soon and progress rapidly; or the disorder previously quiescent suddenly becomes active and soon leads to death. The leukocytes reveal no characteristic changes; occasionally there is a slight leukocytosis (10,000 to 20,000), but rarely any relative lymphocytosis; occasionally an eosinophilia has been observed.

Diagnosis.—Clinically the disease is often not an entity sharply distinguishable from other forms of glandular enlargement. The diagnosis virtually depends upon painless, discrete, progressive enlargement of the lymph nodes unassociated with leukemic changes in the blood; but in the differential diagnosis one must remember that leukemia may have a preleukemic, an aleukemic, and a subleukemic stage. Excision and microscopic examination of an enlarged gland may be the only way to a correct diagnosis in some cases. Some cases of sarcoma (round-cell, spindle-cell, lymphosarcoma) of the lymph nodes often resemble Hodgkin's disease, but, as a rule, in sarcoma growth is rapid and often painful, infiltration and penetration of the capsules of the nodes, fusion of the nodes, and infiltration of the adjacent tissues occur early, metastases are common, and the primary as well as secondary growths may soften, ulcerate, and discharge externally. Sarcoma, however, may be painless and the enlarged nodes of Hodgkin's disease may not remain discrete, so that a differential diagnosis may not be possible in the absence of a microscopic examination of an excised node. Tuberculosis of the lymph nodes rather than Hodgkin's disease is suggested by a tuberculous focus elsewhere in the body, the tendency of the enlarged nodes to fuse, form adhesions with the skin, soften, suppurate, and discharge, and a response to the tuberculin tests; but a microscopic examination of a node or bacteriological examination of the purulent discharge may be necessary to establish the diagnosis. Syphilis may be distinguished by the history and concomitant evidences of syphilis elsewhere in the body. The types of splenomegaly formerly called splenic pseudoleukemia, von Jaksch's disease, etc., and the tumor formation known as multiple myeloma, and formerly called medullary or myelogenous pseudoleukemia, are in no way related to Hodgkin's disease. Multiple myeloma (occurring especially in the ends of the long bones, the ribs, the vertebræ, etc.) is characterized by deep-seated, sometimes pulsatile (bone) pains, bony enlargements, perhaps spon-

taneous fractures, divers motor and sensory phenomena due to the local growths, fever, anemia without leukocytosis (perhaps occasionally relative lymphocytosis), and Bence-Jones albumosuria (so-called myelopathic albumosuria, Kahler's disease).

Prognosis.—The disease is invariably fatal. Most subjects rarely live more than three years; very mild and slowly progressive cases may continue six years or more. Periods of quiescence, with spontaneous regression of the enlarged lymph nodes, apyrexia, and improvement in the general health sometimes occur, and may last for months; or the disease for some time mild and non-progressive suddenly takes on malignant characteristics and speedily leads to death. Death may result from progressive anemia, cachexia, and asthenia; or from tuberculosis or other secondary infection (sometimes answerable for the high fever toward the end).

Treatment.—The nature of the disease makes it especially refractory to treatment. The best results (lessening of the progress of the disease) have been attained by the use of arsenic and the *x*-rays, which should be employed as advised in leukemia. Operative removal of all the regional lymph nodes in the localized form of the disease has rarely been of some temporary avail, but, as a rule, all nodes cannot be removed, and those left soon take on renewed growth.

DISEASES OF THE THORACIC DUCT.

The thoracic duct is formed by the confluence of the main trunk or trunks of the intestinal lymphatics and the main lumbar trunks which have resulted from the union of the lymphatics of the pelvis, genitalia, abdominal wall, and lower extremities. The point of union is on a level with the first or second lumbar vertebra, and is usually indicated by a saccular dilatation—the receptaculum chyli. Thence the thoracic duct courses through the posterior mediastinum, and, being joined by the main trunk of the lymphatics of the upper part of the body (except those of the right side of the head and thorax), eventually empties into the left subclavian vein distal to its union with the internal jugular. The right thoracic (or lymphatic) duct empties into the right subclavian (sometimes internal jugular) vein. Anatomical anomalies, however, are by no means rare.

Pathology.—The thoracic duct is subject to divers disorders of considerable interest in clinical medicine: (1) It is a common source of the rapid dissemination of tubercle bacilli throughout the body—by way of the lymph and blood streams. The duct often acts merely as a passive carrier of bacilli discharged by a tuberculous lymph node somewhere within the area of collection of its tributaries; sometimes it is itself tuberculous. There is considerable evidence also that it is the avenue of general distribution of pyococci in many cases of septicopyemia; and of carcinomatosis developing primarily in the abdomen

or pelvis. (2) It is often the seat of different disorders, of which the ultimate consequence is obstruction. This may arise from thrombi, parasites (Filaria bancrofti), secondary tumor masses, etc., within the duct; acute and chronic inflammation or primary tumor formation of the duct itself; compression of the duct from without by regional inflammatory exudates, cicatrices, enlarged lymph nodes, tumors, aneurysm, etc.; or thrombosis of the subclavian vein. The consequences vary with the site and degree of the obstruction. If the obstruction is incomplete, an efficient collateral circulation may become established and there may be no noteworthy symptoms; this may ensue also when the obstruction is low down. When the obstruction is complete and high up, the duct usually becomes dilated; the receptaculum chyli may become as large as a walnut or a hen's egg (perhaps apparent on palpation of the abdomen). Sometimes the dilatation extends to the peritoneal tributaries of the duct, giving rise to lymph or chyle cysts (so-called chyle angiomas, which, however, often have a local inflammatory peritoneal cause, or resemble tumor formations). In some cases of extreme obstruction rupture of the duct ensues.

Symptoms.—The symptoms are those of stasis of chyle, with the eventual development, in consequence of transudation of the chyle or rupture of the vessels, of chyloperitoneum (chylous, milky, opalescent ascites), and chylothorax; perhaps also chylopericardium, chyluria, and chylous swelling of the abdominal wall, external genitalia, legs, etc. In the event of rupture of the duct rapid emaciation usually ensues.

Diagnosis.—It is sometimes difficult to distinguish chylous from so-called chyliform fluid which (in the absence of obstruction of the lymphatics) sometimes develops in the peritoneum, pleura, etc.; it is due to fatty metamorphosis of leukocytes (pus cells), endothelium, and tumor cells, and has been found in chronic, especially tuberculous, inflammations of the serous membranes, endothelioma, chronic heart disease, cirrhosis of the liver, etc. The presence of fine (rather than coarse) fatty droplets and of sugar suggests, but is not conclusive evidence of, the chylous origin of the fluid; in some cases of chyliform fluid the origin of the fat is evident in the presence of fatty degenerated cells.

Prognosis.—The prognosis is bad, as a rule; but it depends upon the nature and completeness of the obstruction. Occasionally an efficient collateral circulation becomes established, or a thrombus, for instance, may become softened and permit of some circulation through the duct, or the course of the obstruction being removable, may be removed.

Treatment.—The treatment, in cases in which any treatment is of avail, is purely surgical.

DISEASES OF THE THYMUS BODY.

Pathological Physiology.—The thymus body ordinarily attains its greatest development at the end of the second year of life. Thereafter it gradually atrophies until puberty, whereupon the atrophic process

becomes more rapid, so that in adult life the body is represented by a small mass of fibro-adipose tissue containing Hassell's corpuscles (remnants of the original epithelial tissue) and a small amount of lymphoid tissue. Anatomically and functionally the thymus is to be classified with the lymphoid tissues, especially since nothing is known of the function of Hassell's corpuscles, and there is no certain evidence that the thymus produces a specific internal secretion. There is some evidence, however, that an obscure and ill-comprehended relationship exists between the thymus and organs that do produce an internal secretion, such as the thyroid (in Graves' disease), parathyroids, pituitary body (in akromegaly), the ovaries, testicles (atrophying as the sexual organs develop), etc.; and extracts of the thymus are said to cause a fall in blood pressure and rapid action of the heart. Like adenoid tissue generally, the thymus is the seat of lymphocyte formation, especially in early life when they are produced in abundance; furthermore, upon slender basis, the thymus is said to exert some influence upon the growth of the bone and the development of the cerebral functions, and to participate in the processes of immunity. The thymus persists in castrated animals. Apparently the thymus is rarely the seat of primary disease, the only known disorder of clinical significance being enlargement or hypertrophy, in some way concerned in thymic asthma and thymic death.

Enlargement is sometimes found also in leukemia, Hodgkin's disease, rachitis, Graves' disease, Addison's disease, akromegaly, myasthenia gravis, etc., as well as in tumor formations (primary, that is, benign and malignant lymphoma, and secondary), cyst formations, etc. The thymus may be affected secondarily also in a number of disorders, such as acute especially pyococcic infection (acute simple, or suppurative thymitis), chronic infections (tuberculosis, syphilis, etc.). Rarely, pathological atrophy has been encountered in some cases of infantile marasmus, but it is probably part of the general process rather than of etiological significance. Extracts (fresh or dried) of the thymus have been used with varying success in arthritis deformans, rickets, myxœdema, Graves' disease, Addison's disease, etc.

ENLARGEMENT OF THE THYMUS.

(Hypertrophy, Hyperplasia, Persistence of the Thymus; Status Lymphaticus; Lymphatism; Thymic Asthma; Thymic Death; Thymic Tracheostenosis.)

Aside from the disorders already mentioned in which the thymus may be enlarged, enlargement is found in a series of cases characterized by stridorous dyspnœa, asthma, or sudden death. Some authors maintain (although others deny) that the thymic enlargement is the cause of the stridor, asthma, and death which it is believed to provoke by pressure on the trachea (tracheal stenosis), on the vagus, or other of the mediastinal tissues or organs.

The enlargement is a hyperplasia of unknown cause; it has been

referred to divers sorts of infections, intoxications, and disorders of metabolism. Often it is associated with adenoids, enlarged tonsils, enlarged lymph nodes generally, enlarged spleen, and reddish bone marrow (status lymphaticus); sometimes also there is hypoplasia of the heart and aorta, infantilism, etc. There has been much discussion as to whether enlargement of the thymus can cause tracheal stenosis. Warthin, who has studied the subject attentively, maintains that it may; suggesting the use of the term "thymic tracheostenosis," he states that "all the symptoms and all the operative and postmortem evidence point to a suffocation resulting from tracheal stenosis. Secondary laryngeal spasm is the chief if not the only cause of the fatal termination. To these may be added other effects of compression upon the heart, great vessels, vagi, and recurrent nerves. To a reflex spasm of the glottis may be added a reflex cardiac paralysis, as the latter may alone be the direct cause of death in those cases of sudden death in which all signs of tracheal stenosis or laryngeal stenosis are wanting." "Status lymphaticus may be the consequence of a number of primary morbid processes, such as syphilis, rachitis, some latent infection, auto-intoxication, etc., that are characterized by excessive demands upon the lymphoid and myeloid tissues. The sudden death is dependent primarily and wholly upon the thymic enlargement, and the latter condition becomes, therefore, the most important feature clinically." Other observers believe that there is a toxic element in the condition.

Symptoms.—The symptoms are said by Warthin to consist of respiratory disturbances resulting from the diminution of space in the superior thoracic strait. He distinguishes three classes of cases according to their severity—thymic stridor, thymic asthma, and thymic death. The stridor is usually congenital or develops soon after birth; it is inspiratory and expiratory; it may be stationary or progressive, or it may manifest itself suddenly in its most marked form; it may be precipitated by a fit of screaming or crying, or it may come on during one of the acute infections. It varies much in intensity; it is unassociated with changes in the voice, and persists during sleep, although it lessens in severity. Thymic asthma (Kopp's asthma, Millar's asthma) is an exacerbation of stridor of asthmatic character, or it may develop in a child showing no previous sign of stridor. The first attack may be fatal, or there may be periodical recurrences of progressive character; but an attack may subside quickly. There is no sharp line of demarcation between thymic asthma and thymic death, but the term thymic death has come to be applied to those cases in which death occurs suddenly without definite history of previous respiratory difficulty. It may be induced by divers causes, such as anesthesia, slight surgical operations, fright, etc. The general picture is that of a convulsive attack of thymic asthma of severest type ending in death (Warthin).

Physical examination discloses an area of dulness at the upper part of and somewhat to both sides of the sternum, merging with the upper limit of the cardiac dulness. Sometimes there is distinct bulging, or a

palpable swelling in the episternal notch. A shadow may be disclosed by the *x*-rays. Tracheal or bronchial stenosis may be seen by the bronchoscope or after tracheotomy. The child may be rachitic or anemic. Examination of the blood may reveal a relative lymphocytosis.

Diagnosis.—A congenital chronic stridor both inspiratory and expiratory, but most marked on inspiration, with a tendency to exacerbations, and the production of dyspnœa so marked as to occasion intubation and tracheotomy, and finally death, is usually due to thymic enlargement (Warthin).

Prognosis.—The prognosis is bad.

Treatment.—The underlying rickets, anemia, or malnutrition, should be treated, so as to prevent, if possible, the onset of symptoms referable to the enlarged thymus. By avoiding excitement, operations, etc., attacks may be prevented. During the attack the child should be kept horizontal, at least the head forward, cold applications should be applied to the chest or the child should be immersed in a full warm bath, and supportive measures should be used. Intubation with a long tube through the mouth, or through a tracheotomy wound, may be resorted to. The *x*-rays may be of service, as in leukemic conditions, but until more data are accumulated they must be employed with caution. The thymus has been removed in several cases with good results.

DISEASES OF THE SPLEEN.

Pathological Physiology.—The functions of the spleen are ill understood. The organ seems to be non-essential to life, and it apparently does not produce an internal secretion—whence it cannot be classed with the ductless glands. In general it may be said functionally and anatomically to resemble the lymph nodes—which take on a compensatory hyperplasia after splenectomy. During fœtal life the spleen is the seat of erythrocytogenesis, a function to which apparently it may revert in the stress of circumstances in later life. In diseases of the hemopoietic system, especially leukemia, as already mentioned, it undergoes considerable hyperplasia or metaplasia, so that, especially in myelocytic leukemia, it may become converted into a tissue much resembling, almost identical with, the bone marrow. It seems to be actively concerned in hemolysis: perhaps during health its action in this regard is rather passive, the organ acting merely as a scavenger, or as a graveyard for blood corpuscles incapacitated or destroyed elsewhere in the economy; in diseased conditions, however, hemolysis seems to be active in the spleen, and, as in pernicious anemia, this may be a contributory factor in the fatal termination. The spleen also is concerned in the processes of immunity; this is obvious in the splenic phagocytosis, but perhaps the organ exerts also a chemical non-phagocytic immunizing influence.

ANOMALIES OF FORM AND POSITION OF THE SPLEEN.

In association usually with other errors of development, the spleen may be absent, abnormally small, abnormally large, or misplaced in the abdominal or thoracic cavity; it is sometimes unusually lobulated; and small supernumerary or accessory spleens are quite common. Clinical interest attaches to the presence of the spleen in the right side in transposition of the viscera.

Movable spleen (floating spleen, splenoptosis). Rarely, and then only in the event of considerable enlargement, the spleen may be unduly movable without evidences of displacement of the other abdominal organs; but usually splenoptosis is merely part of general visceroptosis.

RUPTURE OF THE SPLEEN.

Rupture of the spleen may occur spontaneously or in consequence of trauma. Severe trauma may cause rupture of a previously normal spleen; but commonly rupture results from slight trauma, although it may occur spontaneously, in a spleen already diseased, such as the acute enlargements of typhoid, relapsing, or malarial fever (especially in the tropics), infarct and abscess, etc. The trauma may consist of coughing, rough handling, as in palpation, puncture by an exploring needle, etc. The *symptoms* consist of local pain and distress, and the shock and other phenomena attending intraperitoneal hemorrhage. The *treatment* is surgical, and should be instituted immediately.

THROMBOSIS, EMBOLISM, AND ABSCESS OF THE SPLEEN.

Thrombosis of the splenic vein rarely develops in the course of typhoid fever or local inflammatory disorders (such as of the pancreas, etc.); it may result from extension of a thrombus in the portal or mesenteric veins, or follow sclerosis and atheroma of the splenic vein. The symptoms consist in the main of venous engorgement, enlargement and perhaps infarction of the spleen, recurring hematemesis, and other manifestations suggesting splenic anemia, although it differs in the more common, acute onset. Embolism develops usually in the course of acute or chronic valvular disease of the heart and septicopyemic processes; it may follow primary splenic arterial or venous thrombosis. The ensuing infarcts may be simple (mechanical) or septic (with ensuing abscess formation). The condition may be suspected when, in the course of the aforementioned diseases, sudden pain develops in the region of the spleen, becomes aggravated upon motion and respiration, and is accompanied by tenderness, especially on palpation; and when examination discloses enlargement of the spleen and perhaps palpable frictions (acute perisplenitis),

which may be audible on auscultation. Irregular chills, fever, sweats, and leukocytosis may be due to the primary infection, or to the development of a splenic abscess.

ENLARGEMENT OF THE SPLEEN.

Acute enlargement of the spleen (*acute splenic tumor*) occurs in many infections, notably typhoid fever, malaria, and relapsing fever, in which it attains considerable diagnostic significance; but it may be present also in septicopyemia, pneumonia, etc.; in acute portal obstruction, in embolism and thrombosis of the splenic vein, etc. In venous obstruction its cause is obvious; in infection it is attributable to a local congestive and inflammatory reaction to the presence of microörganisms or their toxins or to toxic paralysis of the vasomotor nerves. *Symptoms* may be in abeyance, although local pain and discomfort may supervene, and on examination the spleen may be found enlarged and tender.

Chronic enlargement of the spleen (*chronic splenic tumor*) occurs in: (1) Passive congestion due to chronic heart disease, chronic liver disease (especially cirrhosis), pylephlebitis and other obstructive disorders of the portal circulation; (2) disorders of nutrition, such as rickets, amyloidosis, certain obscure (especially tropical) gastro-intestinal intoxications, arthritis deformans, etc.; (3) infections, such as malaria, syphilis, tuberculosis, kala-azar, and other ill-understood, tropical disorders; (4) diseases of the hemopoietic system, such as chlorosis, pernicious anemia, leukemia, erythema, Hodgkin's disease, etc.; (5) tumors and cysts of the spleen; and (6) certain ill-understood diseases discussed under the heading splenomegaly and splenic anemia. The enlargement of the spleen per se may not cause any noteworthy *symptoms*, but local distress and a sense of weight are not uncommon. The enlargement is readily disclosed by physical examination. An enlarged spleen should not be mistaken for a displaced, though otherwise normal, spleen.

SPLENOMEGALY; SPLENIC ANEMIA.

In a considerable number of more or less ill-understood disorders enlargement of the spleen constitutes a noteworthy feature. The imperfect state of our knowledge precludes a satisfactory classification, but several groups of cases may be distinguished:

1. **Congenital or Familial Splenomegaly, with Acholuric Jaundice** (**Minkowski**). This is usually a familial disorder, and may occur in succeeding generations. The splenomegaly is often extreme; there is slight jaundice, but the stools are not clay-colored, and the urine does not contain bile pigments, although it does contain urobilin and urobilinogen. The blood reveals the changes of secondary anemia. Necropsy has revealed chronic hyperplasia of the spleen without cirrhosis

of the liver or angiocholitis. The cases must be distinguished from those of congenital jaundice in which angiocholitis or obliteration of the biliary ducts has been found. The general health often remains notably unimpaired.

2. **Congenital or Familial Splenomegaly, with Marked Constitutional Disturbances**—stunted growth, infantilism, osseous changes, clubbed fingers, pigmented skin, enlarged liver, and sometimes jaundice. The cases have been encountered especially in children and young adults. Whether there is any relationship between these cases and the Minkowski type of acholuric splenomegaly and Hanot's biliary cirrhosis of the liver remains to be determined.

3. **Primitive Splenomegaly.** This comprises a series of cases characterized by progressive enlargement of the spleen occurring without known cause. In some cases, for many (ten to twenty) years, the general health may be scarcely, if at all, impaired, the enlargement of the spleen constituting the only noteworthy feature of the disease (simple splenomegaly). The spleen may reach extraordinary dimensions, extending to the right beyond the median line, and far into the pelvis. As a rule, however, at the end of three or four years, more or less anemia (splenic anemia) supervenes, perhaps attended or preceded by hemorrhages (usually hematemesis, but sometimes enterorrhagia, hematuria, epistaxis, menorrhagia, purpura, etc.). The erythrocytes, as a rule, vary from 3,500,000 to 4,000,000, the color index is low (0.5 to 0.7), and the leukocytes are diminished. With the progress of the disease and sometimes in consequence of recurring hemorrhages, the anemia becomes more marked, and, reaching a high grade, may lead to death; exacerbations, as in pernicious anemia, are not uncommon. In some cases after the lapse of years, signs of portal cirrhosis of the liver (ascites, etc.), associated usually with slight jaundice, develop (Banti's disease). The conspicuous lesions found in these cases of splenic anemia consist of chronic connective tissue hyperplasia of the spleen, leading gradually to atrophy of the lymphoid tissue, extraordinary hyperplasia of the endothelial cells of the splenic sinuses, and endothelial proliferation and sclerosis (even atheroma) of the splenic vein; sometimes similar lesions involve also the portal vein, both without and within the liver, and lead to stenosis and thrombosis of the branches of the splenic and the portal veins. In some cases the proliferation of the endothelium of the splenic sinuses constitutes the conspicuous feature of the disease, and has been, though incorrectly, interpreted by some observers as related to, if not identical with, a tumor formation (splenic anemia or primary splenomegaly of the Gaucher type; primitive endothelioma of the spleen); identical changes may be found also in the liver, lymph nodes, and bone marrow. The nature of the disorder is not known, although the splenomegaly is assumed to be the primary process; perhaps it develops in consequence of some infective or toxic process resident in the spleen, or to the influence of which the spleen may be unduly susceptible. This toxin may provoke the endothelial and other proliferations and ultimately lead to the

anemia and the hepatic cirrhosis. The undoubted influence of the spleen is evident in the prompt recovery and maintenance of health that often succeed removal of the spleen before the advent of unalterable changes in the liver.

4. **Anemia in Children.** In many of the secondary anemias of infants and children, such as those due to rickets, syphilis, poor food, faulty nutrition, general bad hygienic conditions, etc., as well as the more common causes that provoke anemia in adults, the spleen is likely to be, although it is not always, enlarged; the enlargement varies much in different cases. The etiological factor should be sufficiently obvious; the blood changes are those of ordinary secondary anemia, although a slight leukocytosis is not uncommon; the condition need not be further discussed. There are, however, other cases of anemia that develop in children without obvious cause and are associated with more or less, sometimes considerable, enlargement of the spleen; they are sometimes spoken of as the splenic anemias of children. These cases are difficult to classify: some observers believe that the primary disturbance is in the spleen (the disease being, therefore, akin to the splenic anemia of adults); others that the splenomegaly is the result of or due to the anemia; others that both the anemia and the splenomegaly are the result of a common causative factor (perhaps these are allied to pernicious anemia of adults); and others that some at least of the cases are leukemia or Hodgkin's disease modified in its clinical and anatomical features by the young, more or less undifferentiated, or unduly susceptible, hemopoietic tissues of infants and children. Several grades of the disorder (or disorders) may be distinguished: (1) That in which the blood picture is that common to secondary anemias (diminished erythrocytes with low color index) and a normal number of leukocytes, with perhaps a relative lymphocytosis; (2) that differing from the first mentioned only in the presence of a moderate leukocytosis (up to 20,000), with little if any disturbance in the percentage of the different forms of leukocytes; and (3) that characterized by more marked anemia (3,000,000 or less erythrocytes), a variable, sometimes low, sometimes rather high, color index, considerable leukocytosis (20,000 to 50,000 or more), poikilocytosis, anisocytosis, polychromatophilia, megalocytes, megaloblasts, and a variable number of myelocytes (20 to 40 per cent. or more), including so-called stimulation forms—the disorder formerly called anemia infantum pseudoleukæmica of von Jaksch. These three grades doubtless often, if not always, represent stages or degrees of the one process, and although the causative factor cannot be determined, it is not unlikely that some of the cases are due to the sometimes more obtrusive causes of ordinary secondary anemia. Recovery may ensue, even when the blood contains a large number of myelocytes. The *treatment* is that of chronic secondary anemia.

TUMORS AND CYSTS OF THE SPLEEN.

Tumors of the spleen may be benign (angioma, fibroma, lymphadenoma) or malignant (sarcoma, endothelioma, carcinoma), and primary or secondary. The *symptoms* consist of local pain and tenderness, enlargement of the organ, which may reveal nodules, and the anemia, emaciation, and cachexia common to tumors in general. The *diagnosis* is rarely made before operation (for enlargement), or the necropsy. Cysts of the spleen may be simple (serous, hemorrhagic, lymphatic), dermoid, or parasitic (echinococcus usually). When large, the cyst may cause local distress and reveal itself as a large perhaps fluctuating mass. The *diagnosis* rarely is more than a hazard.

SECTION VI.

DISEASES OF THE CIRCULATORY SYSTEM.

Pathological Physiology.—Studies by Gaskell, Keith, Mackenzie, Wenckebach, Hering, His, Erlanger, and others have tended to emphasize the importance of the myogenic as contrasted with the neurogenic theory of the cardiac action; and although perhaps the two theories may yet be found to possess something of truth, the five functions of the heart muscle demonstrated by Gaskell and Engelmann constitute the basis of the modern conception of cardiac physiology and pathology. These are: (1) Stimulus production, the power of producing stimuli that excite the heart to contraction; (2) excitability, the power of responding to stimuli to contraction; (3) conductivity, the power of transmitting the stimulus from one part of the heart to another; (4) contractility, the power of contracting in response to stimuli; and (5) tonicity, the power of maintaining such contraction of the heart muscle that the total volume of the heart and the size of the cavities are less than they would be were full relaxation of the muscle to occur during diastole.

The power of stimulus production seems to be inherent in the muscle cells of the heart. The nature of the stimulus is not known, but its production is much dependent upon the presence of ionized salts of sodium, potassium, and calcium. The stimulus seems to be, as it were, a product secreted by the cells and stored until sufficient has been produced to excite a contraction. The contraction of the heart depends upon its excitability, which disappears immediately the heart has contracted; the muscle fibers are said to be in the refractory stage. This subsides, that is, excitability returns, during diastole, so that, in general, the longer the period of rest the weaker the stimulus that may excite contraction. The normal rate and rhythm of the heart are dependent upon the normal interaction of the two functions of stimulus production and excitability. In disease the excitability may be increased or diminished. Normally the stimulus to contraction arises in the remains of the sinus venosus, the sino-auricular node (of Keith and Flack) at the mouth of the superior vena cava (the most excitable portion of the remains of the primitive cardiac tube), and it is transmitted by the muscle fibers (perhaps by the bundle of Thorel) along the auricle to the auriculoventricular bundle of His, and thence to the ventricles. The regular succession of contractions of the sinus, auricle,

and ventricle constitutes the normal or sinus rhythm of the heart. In disease remains of the primitive cardiac tube other than the sino-auricular node, becoming unduly excitable, may inaugurate contractions (extrasystoles) independently of the sinus rhythm, and the conductivity, especially of the auriculoventricular bundle, may become depressed (heart block). The power of contractility, the most obvious of the heart actions, is peculiar in that each contraction is maximal and is followed by a long refractory period. The fact of the contraction being maximal, that is, as powerful as the heart is at the time capable of, renders the heart independent of the strength of the stimulus, insures a forceful response (contraction) to a weak stimulus, and prevents an excessive response to a strong stimulus; the refractory period insures a period of rest and renders tetanization of the muscle impossible. In general the strength of the contraction is proportional to the duration of the immediately preceding period of rest. The power of contractility may become much depressed in disease. Tonicity is a property possessed by the cardiac musculature in common with muscular tissue elsewhere in the body; it keeps the volume of the heart and the size of the cavities somewhat less than they would be were full relaxation of the muscle to occur during diastole. It is the force, therefore, that opposes dilatation; it varies much in different individuals in health and disease; it may be increased by drugs, such as digitalis; and it may be diminished by drugs, such as muscarin, and by the toxins of infectious diseases. Depression of tonicity is the obtrusive manifestation of failure of the heart. The coördinated activity of all the functions of the heart is essential to the maintenance of the efficiency of the circulation, and this coördinated activity in turn depends upon a sufficiency of nutriment.

In studying diseases of the heart and interpreting their manifestations, it is essential to have in mind not only the myocardium and its nervous control exerted by way of the pneumogastric and the accelerator nerves, but also its vascular or vasomotor control exerted through the elasticity and contractility of the bloodvessels. This is of importance because of its bearing upon the blood pressure, which is the resultant of the resistance in the arterioles (controlled by the vasomotor nerves), of the elasticity of the arterial walls, of the force of the heart beat, and of the volume of blood. Without discussing the subject at length, it perhaps suffices to point out that the circulation may fail at the periphery as well as at the heart, and that this is more often the case than is usually supposed—as in shock generally, in the heart failure (more properly vasomotor failure) of many infectious processes, etc. Furthermore the beginnings of serious cardiovascular disease are often in the periphery; here divers endogenous and exogenous poisons exert their deleterious influence, and in consequence of a direct irritant action on the vasomotor nerves, or perhaps on the muscular coat of the vessels, increase the blood pressure, and inaugurate the series of changes discussed under arteriosclerosis. It is essential, therefore, to study the arterial, the capillary, the venous, and the pulmonary circulation.

In all diseases of the heart the major factor to be determined is the functional capacity of the heart, in comparison with which other matters are of minor significance. Noteworthy incapacity of the heart is quite obvious in symptoms of unstable compensation or of impending or actual decompensation; of these, several, such as cardiac pain, palpitation, irregularities of rate and rhythm, etc., are discussed under the general heading of neuromuscular disorders. There are several others, common to the different forms of heart disease, such as dyspnœa, cyanosis, and œdema, that plainly disclose the functional incapacity, the functional failure, of the heart.

Dyspnœa is very common; it may be the only symptom, but is often accompanied by a sense of precordial or substernal distress or actual pain, and more or less anxiety and apprehension; and it varies much in degree from slight breathlessness on exertion or excitement to orthopnœa even when the patient rests in bed. This dyspnœa is due directly to deficient oxygenation of the blood—to an excess of carbon dioxide, which in turn results from a slow and feeble circulation; the excess of carbon dioxide acts as an irritant to the respiratory centre in the medulla and increases the respiratory rate. Contributing factors are often present, such as chronic passive congestion with resultant changes in the alveolar epithelium and encroachment upon the interalveolar space in consequence of dilatation of the capillaries, loss of elasticity of the lungs, œdema of the lungs, chronic bronchitis, bronchopneumonia, etc. The dyspnœa sometimes comes on paroxysmally in attacks—so-called cardiac asthma. These attacks are due to temporary incapacity of the left ventricle, which may be provoked by exertion, excitement, a full meal, flatulence, constipation, etc., or the attacks may come on without obvious cause. They are quite common early in the night, developing soon after the patient has fallen asleep. Sometimes, especially in arteriosclerotic subjects (cardiovascular-renal disease) with involvement of the brain, the respirations are peculiarly periodical, longer or shorter periods of apnœa alternating with periods of breathing, at first slow and shallow, but gradually becoming deeper and more rapid until they reach a maximum, whereupon they gradually decrease and finally again cease (Cheyne-Stokes breathing).

Cyanosis attends dyspnœa and is due to like causes—to an excess of carbon dioxide in the blood. In chronic cases the patients not infrequently become more or less accustomed to the cyanosis, the organism adapts itself to the changed conditions, and although the cyanosis may be more or less obvious, there may be little or no subjective dyspnœa, and the functions of the body may be fairly well performed. Cyanosis occurs not only in diseases of the heart, but also in diseases of the lungs interfering with proper oxygenation of the blood; in erythremia (polycythemia with cyanosis and enlarged spleen); rarely in conditions due to alteration of the blood pigment itself: that is, the conversion of the hemoglobin into metahemoglobin (chronic poisoning by acetanilide,

phenacetin, sulphonal, trional, etc.); and in so-called enterogenous cyanosis (intestinal auto-intoxication), due in one series of cases to conversion of the hemoglobin into methemoglobin, the result of the absorption of nitrites from the intestine in chronic diarrhœa, and amenable to a milk diet, and due in another series of cases to conversion of the hemoglobin into sulph-hemoglobin, the result apparently of putrefaction of the proteins in chronic constipation (increased formation or absorption of H_2S), and amenable to treatment directed toward the constipation.

The œdema of cardiac disease is largely the result of slowing of the circulation and consequent stasis of the blood; but changes in the endothelium of the capillary blood vessels and lymph spaces are a contributing factor. This œdema from stasis is to be distinguished from so-called inflammatory œdema, nephritic œdema, and hydremic œdema.

In many cases of readily recognized heart disease, compensation has not failed, nor is it unstable; but it is nevertheless desirable to ascertain the functional capacity of the heart. This may be roughly determined by noting the amount of work or excercise that provokes dyspnœa, palpitation of the heart, precordial distress, and increased frequency of the heart beat; but this is a rude test. Other more or less accurate and satisfactory methods of determining the functional capacity of the heart have been suggested. The graphic methods, especially emphasized by Mackenzie, whereby it is possible to recognize disturbances of excitability, conductivity, contractility, tonicity, and rhythmicity, are excellent; but are not often available in clinical work outside a hospital. Other methods are based upon the changes in blood pressure and pulse rate provoked by a measured amount of work. A simple test consists in determining the difference between the rate of the heart beat in the erect and in the recumbent posture; normally recumbency produces a lessening of the heart beats of from six to twenty per minute; this is partly or completely lost in cardiac insufficiency. Mendelsohn has suggested that the functional capacity of the heart may be estimated by ascertaining the time required for the heart to regain its normal rate after a measured amount of work. Katzenstein determines the blood pressure and the pulse rate after digital compression of both femoral arteries. In normal individuals this compression results in an increase of the systolic blood pressure in the brachial artery of from 5 to 15 mm. Hg., without noteworthy changes in the pulse rate; in cases of efficient hypertrophy the blood pressure may be increased 15 to 40 mm. Hg.; in cases of slight cardiac insufficiency, the pressure may not rise at all, while the pulse rate becomes accelerated; and in cases of marked cardiac insufficiency the blood pressure may fall and the pulse rate become accelerated. The pain and mental excitement induced by the procedure seriously compromise the results, and a marked increase in blood pressure is not entirely free from danger. Gräupner has drawn attention to the fact that after a measured amount of work the systolic blood pressure and the pulse rate thus increased, gradually

return to the normal, and that after the pulse rate has declined to the normal the systolic blood pressure begins to rise, attains its maximum several minutes later than the pulse rate, and then falls to (or below) the normal; this so-called normal recovery may be delayed and diminished in slight cardiac inadequacy, and it may be entirely absent in marked cardiac insufficiency, the blood pressure declining continuously, and only reaching the normal after a longer or shorter period of rest. Stair climbing or lifting weights, may be resorted to for the measured amount of work.

NEUROMUSCULAR DISORDERS OF THE HEART.

Disturbances in the sensory or motor activities of the heart often either dominate the clinical picture or constitute the sole discoverable evidence of cardiac disease. These disturbances, in consequence, are often spoken of as neuroses or functional disorders of the heart, terms that have some justification in fact, but are often misnomers, since the disorders in question often constitute mere symptoms or symptom-complexes and have an anatomical basis. Convenience, however, and the grouping of the phenomena suggest their discussion together under the designation neuromuscular or symptomatic disorders. These may be divided into the sensory and the motor. The sensory disorders are represented by cardiac pain and distress and the subjective sensations associated with palpitation and with some forms of irregularity or intermission of the heart; the motor disorders include disturbances of rate (rapid, infrequent; tachycardia, bradycardia), of rhythm (the different types of arrhythmia), and of force (strong and weak).

Cardiac Pain.—Cardiac pain varies much in character, intensity, and in situation. It may be a mere discomfort, or an actual pain—dull, aching, stabbing, or agonizing, griping, constricting, vise-like. It may be referred to the apex of the heart, to the entire precordium, to the axillæ, and it may radiate to the back, to one or both arms, etc. The agonizing, constricting, vise-like pain of angina pectoris is, of course, the obtrusive manifestation of the disorder; but since the disorder has an anatomical basis, it should not be called a neurosis.

Cardiac pain may be due to organic or so-called functional disorders of the heart: (1) The organic cases comprise all the diseases of the myocardium, endocardium, and pericardium, and of the aorta, that will be discussed under the respective headings. (2) Other cases are due to the toxic action of tobacco, alcohol, tea, coffee, etc., and may or may not be associated with demonstrable organic changes; the pain or distress usually ceases in the course of time upon discontinuance of the exciting factor. (3) A third group of cases are the so-called nervous cases; cases induced by shock, emotional disturbances, excessive venery, etc.; or associated with neurasthenia, hysteria, disorder of the genitalia (male and female) or of the kidneys, etc.; or apparently due to disorder

of the vasomotor system (Nothnagel's vasomotor angina). (4) Some cases are due solely to gastro-intestinal derangements associated with flatulence; they are readily cured by attention to the exciting cause.

Palpitation of the Heart.—In health the activity of one's heart is entirely unnoticed; the subjective consciousness of the heart's pulsation, so-called palpitation, develops under a variety of circumstances. It is a normal phenomena under the stress of unusual exertion or excitement, but the sensations of throbbing thus experienced are perfectly regular, as a rule, and soon subside with the initiating cause. It develops upon slight, sometimes very slight, provocation in cases of unusual excitability of the nervous system, in neurasthenia and hysteria; and in women generally rather than in men, especially at times of nervous perturbation, as at puberty, during menstruation, at the climacteric, and in conditions of worry, anxiety, and emotional disturbances. It occurs also in consequence of the toxic influence of tobacco, alcohol, tea, coffee, etc., on the heart; in the myocardial degenerations associated with anemic conditions and the acute infections; in the condition of irritable heart, due to excessive muscular strain; in organic (myocardial and endocardial) diseases of the heart generally; and in many disorders of the abdominal organs, notably flatulent dyspepsia.

The attacks vary much in severity—from a slight and transitory feeling of fluttering of the heart to severe spells of very forcible throbbing of the heart, attended by marked precordial distress or oppression, that may continue for an hour or more. The throbbing not infrequently extends to the arteries and may lead to flushing of the skin and perhaps to perspiration and transient polyuria. Usually the heart's action is much accelerated, up to 150 or more per minute, and often irregular; but occasionally the rate and rhythm are quite normal; and on the contrary some attacks of very rapid heart action (delirium cordis) may be entirely unperceived by the patient. Examination of the heart, as a rule, reveals only the evidence of the hyperactivity. The sounds usually are normal or perhaps somewhat accentuated, especially the pulmonary second sound. There may be a slight systolic murmur in the pulmonary area, especially in the anemic and neurasthenic cases.

Arrhythmia.—The following varieties of arrhythmia may be distinguished:

1. **Respiratory Arrhythmia (Juvenile Arrhythmia; Sinus Irregularity Mackenzie).**—Respiratory arrhythmia is an exaggeration of the normal inspiratory increase and expiratory decrease in the frequency of the cardiac action; it is characterized by variations in the duration of the cardiac cycle, dependent almost if not quite wholly upon prolongation of the diastole. It is not associated with noteworthy changes in the size of the individual pulse beats, nor is it a condition of missed beats at the wrist due to extrasystoles. It is not uncommon in youths approaching puberty, but rarely occurs in healthy adults; it is sometimes observed during convalescence from acute infections (diphtheria, scarlet fever, typhoid fever, pneumonia, etc.), in anemic conditions, in neurasthenia, in cerebral disorders, tuber-

culous meningitis, etc. It is often associated with slowing of the heart action and infrequent respiration, and is attributable to irritation of the vagus (exaggeration of the normal respiratory reflex). It is readily recognized: beats of equal strength varying continually in time with the phases of respiration, being, as a rule, most infrequent during expiration. Occasionally in children it may be mistaken for continuous irregularity (nodal rhythm), but it may be distinguished by a tracing of the jugular vein which shows concurrent involvement of the auricle, and by its being lessened or dissipated by atropine (which does not influence continuous irregularity), and by being increased by digitalis (which lessens continuous irregularity). It should not be confused with the parodoxic pulse—inspiratory diminution in the force and volume of the pulse. Of itself it is of minor significance and good prognosis, and it calls for no special treatment.

2. **Extrasystoles.**—The majority of cases of arrhythmia are due to extrasystoles, that is, to "premature contractions of the auricle or the ventricle in response to a stimulus from some abnormal point of the heart, but in which otherwise the fundamental or sinus rhythm of the heart is maintained" (Mackenzie). Mackenzie reasons that, since the stimulus for contraction arises normally in the remains of the sinus venosus at the mouths of the great veins (the sino-auricular node), other remains of the primitive cardiac tube, if rendered more excitable, might start the contraction before the sinus; and since this primitive cardiac tube in the heart is represented also in the auricle, in the auriculoventricular node, and in the auriculoventricular bundle after it leaves the node, he (as well as others) describe the following types of extrasystoles:

1. VENTRICULAR EXTRASYSTOLES, those in which the origin of the extrasystole is assumed to be in the auriculoventricular bundle beyond the auriculoventricular node (that is, in the ventricle). This stimulates the ventricle to contraction in advance of the normal time; this contraction (extrasystole) is followed by the usual refractory period which extends beyond the time that the normal stimulus from the sino-auricular node reaches the ventricle; the ventricle in consequence remains inactive, a beat is missed, and a large so-called compensatory pause ensues, which is terminated by a forcible (maximal) contraction of the ventricle in response to the next ensuing stimulus from the auricle; this occurs at the proper time and in normal rhythm. Characteristic of the ventricular extrasystole is the fact that the time occupied by the normal systole, the extrasystole, and the compensatory pause is twice that of a normal cardiac cycle; the number of heart beats per minute may remain normal, a premature ventricular systole (extra-systole) being interpolated between two normal contractions.

2. AURICULAR EXTRASYSTOLES, those in which the stimulus is assumed to arise in the remains of the primitive cardiac tube incorporated in the auricle elsewhere than at the sinus. As in the ventricular extrasystoles, the premature contraction of the auricle (extrasystole) is followed by a con-

traction of the ventricle; but the normal stimulus from the sinus finds the auricle refractory and contraction does not take place. This form of arrhythmia resembles ventricular extrasystoles, and usually is distinguishable only by tracings of the jugular pulse (which reveal the auricular extrasystole). As in ventricular extrasystoles the time occupied by the normal systole, the extrasystole, and the long pause may be twice that of a normal cardiac cycle; but in most cases it is less; that is, the pause is not compensatory.

3. Auriculoventricular extrasystoles (nodal extrasystoles), those in which the stimulus is supposed to arise in the auriculoventricular node of Tawara, and gives rise to premature and simultaneous contractions of auricles and ventricles. This is assumed by some observers to be a so-called retrograde extrasystole, that is, a ventricular extrasystole in which the stimulus has travelled back along the auriculoventricular bundle and prematurely stimulated the auricle, an assumption that Mackenzie believes untenable.

Extrasystoles result from the increased excitability of the remains of the primitive cardiac tube (Mackenzie); perhaps occasionally from an exaggeration of the normal automatism of the heart. Mackenzie believes that the compensatory pause is not due to the ventricle being refractory, but to the auriculoventricular bundle being refractory (impaired conductivity), and not conveying the stimulus to the ventricle, which, therefore, stands still until the next physiological stimulus comes down from the auricle. Clinically this type of arrhythmia is characterized by the occurrence of two or more approximated pulse beats followed by an unusually long pause (intermission of the pulse). The extrasystole may occur only at long, irregular intervals, at short irregular intervals, or at short regular intervals, as after every second beat, every three beats, etc., in which latter event it gives rise to the so-called bigeminal, trigeminal, quadrigeminal, quinquegeminal pulse, etc. A continuous series of extrasystoles may give rise to a type of paroxysmal tachycardia (usually, however, this is of the nodal rhythm). In some cases the extrasystole is more or less ineffectual, because it occurs early in diastole (in the refractory period, when the ventricle is incapable of much contractile effort), the ventricle contains very little blood, and the pressure in the aorta is very high—so that the aortic leaflets are opened slightly, if at all. This more or less ineffectual extrasystole produces little if any perceptible influence on the pulse (although it may be registrable on the sphygmogram or cardiogram); the pulse then appears to be definitely intermittent—a beat is missed. If this occurs with regularity after each normal systole of the ventricle, the condition at first may be mistaken for bradycardia, which, however, can be differentiated by tracings of the jugular pulse or of the apex beat, and by auscultation. There is some doubt as to the occurrence of hemisystole (alternate contraction of the two ventricles). Mackenzie and Hering believe that most reported cases are really cases of extrasystole, and that hemisystole occurs only in a dying heart; von Leyden, Kraus, and Nicolai believe, however, that rarely the ventricles may act

asynchronously. On auscultation the extrasystole, if it occurs early in diastole and is ineffectual in opening the aortic valve, is revealed by a single, usually a muffled, sometimes a loud, sound; if it occurs later in diastole, and does lead to opening of the aortic valve, a second sound may also be produced; the two normal sounds are quickly followed by two weaker sounds and an unusually long pause. Subjects are sometimes conscious of the extrasystole—a dull thud in the region of the precordium, or a sensation of stoppage of the heart and a palpable intermission of the pulse. Although, as a rule, readily recognizable, it is sometimes difficult to distinguish extrasystoles; the several types may occur in varying combinations, and they may be associated with other forms of arrhythmia.

Extrasystoles occur in a variety of conditions: (1) In neurotic subjects, especially after exertion and excitement, at puberty, and the climacteric, during pregnancy, in hysteria, neurasthenia, etc., and reflexly in indigestion and divers gastro-intestinal disorders; (2) in toxic conditions such as those due to tobacco, tea, coffee, alcohol, etc.; (3) in infections, especially during convalescence from typhoid fever, pneumonia, diphtheria, scarlet fever, etc.; and (4) in the various sorts of acute and chronic diseases of the heart, especially those associated with hypertension. In themselves, extrasystoles are of minor clinical significance, of which fact the patient, if aware of the irregularity, should be assured; the associated conditions, such as infections and various sorts of heart disease, may be of great significance, even dangerous. The treatment should be directed to the associated or underlying factors.

3. **Continuous Irregularity (Perpetual Irregularity; Nodal Rhythm; Rhythm of Auricular Paralysis; Ventricular Rhythm; Absolutely Irregular Heart).**—This is characterized by irregularity in the force, equality, and sequence of the pulse beats, by the absence of the fundamental or sinus rhythm of the heart, by the presence of a ventricular (or positive) venous pulse (tricuspid insufficiency or auricular paralysis), by the absence of an auricular wave on the jugular tracing, and by the permanence of the phenomena. The cardiac action may be extremely rapid (delirium cordis); occasionally it may be very rapid temporarily (paroxysmal tachycardia); rarely it may be quite slow, especially under the influence of digitalis; except when very rapid, the cardiac action is always irregular and this may be of minor or of high grade. The auscultatory phenomena are similarly irregular: the sounds vary in character and intensity, many of the systoles are not followed by second sounds, and they occasion no palpable pulse at the wrist.

The condition is due to the concurrent contraction of the auricle and the ventricle (sometimes antecedent contraction of the ventricle), in response to stimuli arising at the auriculoventricular node (Mackenzie), or to a block between the sinus and the auricle (depression of conductivity, Wenckeback, Erlanger). It is found in all forms of severe heart failure with dilatation of the auricles, particularly those of rheumatic origin (mitral stenosis especially) and the fibroid heart. Mackenzie

believes that in a great many cases the immediate breakdown is directly attributable to the inception by the heart of the abnormal rhythm. The condition is always of serious moment. When once established it usually continues until death, although it may be improved by appropriate treatment, especially the use of digitalis; that is, some normal beats occur and irregular beats become less apparent; but evidence of auricular contractions usually do not again become evident, and slight exertion sets up extreme irregularity and rapid action of the heart.

4. **Partial and Complete Heart Block (Depression of Conductivity).**—In some cases, the ventricle fails to contract after each auricular contraction, in consequence of failure of the auriculoventricular bundle of His to conduct the stimulus to the ventricle; the condition, therefore, is a manifestation of depressed conductivity, and is spoken of as partial or complete heart block. But depression of conductivity may occur without arrhythmia. Erlanger has shown experimentally that varying degrees of compression result first in increase of the intersystolic period (*a-c* interval; that is, the time between the beginning of the auricular and the carotid waves in tracings of the jugular pulse); then in a 2 to 1, 3 to 1, or 4 to 1 auriculoventricular rhythm (incomplete heart block); and finally in independent auricular and ventricular contractions (complete heart block). Mackenzie states that the stimulus to the ventricle may be, (1) delayed; (2) prevented from crossing the bundle of His; or (3) completely blocked beyond the auriculoventricular node, in which event the ventricle contracts in response to a stimulus that arises in the uninjured remains of the auriculoventricular bundle (heart block, ventricular rhythm). Clinically the condition is manifested by a slow ventricular rate (slow radial pulse; one type of bradycardia), associated with more frequent pulsations of the veins of the neck, owing to preservation of the normal rate of auricular contractions. If the ventricular contractions are more than thirty-six per minute, they may have a distinct relationship to the auricular waves in the jugular pulse, the ventricle responding to every second, third, or fourth auricular contraction; when the ventricular contractions are about thirty or less, they are probably independent of the auricle, and the auricle and the ventricle beat at independent rates, in response to independent stimuli. The condition may be suspected when there is a complete pause in the radial pulse with absence of heart sounds (Mackenzie).

Temporary and incomplete heart block occurs rarely in certain infections (or during convalescence), such as typhoid fever, pneumonia, diphtheria, scarlatina, rheumatism, influenza, etc.; it may occur in the fibroid heart; and it may be set up by the prolonged use of digitalis.

Complete heart block is typically represented in the so-called Adams-Stokes syndrome, of which the characteristic features are: (1) Bradycardia, sometimes permanent, sometimes paroxysmal and temporary, the radial pulse and the ventricular systoles falling to 40, 20, 10 or less, a minute; (2) visible auricular pulsation of the veins of the neck due to auricular contractions, and often revealing a 2 to 1, 3 to 1, or 4 to 1

rhythm as compared with the ventricular contractions; and (3) cerebral (anemic) disorders—vertigo, syncope, pseudo-apoplectiform attacks, sometimes associated with convulsions (epileptiform) of variable severity and duration. The cases of heart block that have been carefully studied after death have revealed more or less destructive lesions, especially gummas, sclerosis, atheroma, anemic necrosis, etc., of the auriculoventricular bundle of His, sufficient partly or completely to interrupt the conduction of impulses. It is surmised that the less degrees of heart block are due to less-marked, in some cases perhaps merely functional lesions of the bundle. The cases due to digitalis are the result of a specific depressive action of the drug on the function of conductivity. The Adams-Stokes syndrome is a serious malady, ultimately fatal, although it may last for years. The use of the iodides often leads to marked improvement, especially in the syphilitic (gummatous) cases. Strychnine also has proved of avail.

5. **Alternating Pulse (Depression of Contractility).**—The alternating pulse consists of a regular succession of large and small pulse waves, and results from depression of the function of contractility. Mackenzie states that "when contractility is depressed, if time be allowed for a full and strong contraction, the longer duration of contraction encroaches upon the period of rest, so that by the time the next stimulus arrives the contractility has not sufficiently recovered, and a smaller and shorter contraction results. As this contraction is shorter in duration, the period of rest is thereby lengthened before the next stimulus arrives, so that the contraction will be stronger and longer; being longer, it will again encroach upon the period of rest, and so the process of alternation goes on." This alternating pulse is found in conditions of exhaustion of the myocardium, especially in advanced mitral stenosis, coronary sclerosis, general arteriosclerosis with high blood pressure, angina pectoris, fibroid heart, etc. It is of serious moment.

Tachycardia.—The term tachycardia is more or less loosely applied to abnormal frequency of the heart action; by some observers the term is restricted to extraordinary frequency (twice the normal—150 or more); and it is not infrequently confused with the term paroxysmal tachycardia, which seems to be a distinct clinical entity. Abnormal frequency of the pulse may be physiological or pathological. The physiological cases comprise those normal individuals in whom the pulse is persistently above the average of normal persons—80 to 100 per minute. The pathological cases comprise those due to: (1) Infections, which include almost all the infectious diseases, in which, associated with fever, the pulse becomes accelerated. In perhaps the majority of cases the acceleration of the pulse is proportionate to the increment of fever; in some cases, notably scarlatina, the pulse is disproportionately rapid; while in others such as typhoid fever, certain forms of meningitis, although it may be of increased frequency, this is not up to the average of other fever states; in some of these cases the pulse may even be slowed. (2) Intoxications, such as tobacco, tea, coffee, atropine, strychnine, alcohol, etc. (3) All

acute and chronic diseases of the heart, such as irritation, overstrain, valvular diseases (notably mitral stenosis), myocardial disease, pericardial disease, etc., especially in impending or actual decompensation. (4) Neurotic disturbances, such as emotional and other shocks, hysteria, neurasthenia, and some organic nervous diseases. (5) Graves' disease. (6) Paroxysmal tachycardia.

Paroxysmal tachycardia is a disorder characterized by paroxysms of extraordinary frequency of the heart beat and the pulse. It occurs in both sexes, especially in neurotic individuals, and in subjects with previously normal hearts, as well as in cardiopaths. It commonly develops in adult life, although it may come on during childhood. It is usually attributed to paralysis of the inhibiting centre (vagus), but minor grades (a pulse rate of less than 120) are sometimes ascribed to irritation of the sympathetic. Mackenzie regards the condition as a manifestation of extrasystoles (transient and recurrent nodal rhythm). The attacks come on suddenly, usually without obvious cause, last several minutes, several hours, or several days, and usually end abruptly; they may recur at irregular intervals, days, weeks, months, or years; or perhaps there may be only the one attack. The attack is characterized by subjective sensations of oppression, tightness, or fluttering in the region of the heart, associated with restlessness and weakness, and often dyspnœa on exertion. The pulse is found to be small and rapid, often 200 or more per minute (sometimes so rapid that it cannot be counted); often there is no noteworthy inequality in the force, rhythm, or volume of the beats, but more or less (even marked) arrhythmia may develop. Auscultation reveals the heart sounds to be short and sharp (embryocardia; tic-tac), and often a systolic murmur (mitral insufficiency). The heart may become markedly dilated, even within a short time (according to Martius, the disorder is a recurring acute dilatation of the heart), and the veins of the neck and the liver may become distended and pulsate. The *prognosis* depends upon the functional capacity of the heart under the abnormal rhythm, upon the general circulatory conditions under the new circumstances. Dyspnœa and œdema are of bad prognostic significance. Mackenzie believes that many cases of rapid and fatal breakdown, particularly in old people, are due directly to the inception by the heart of this rhythm. The *treatment* comprises bodily rest and mental quietude, under which some attacks subside spontaneously. The bromides, morphine, valerian, and other sedatives may prove of service; belladonna, the iodides, nitroglycerin, etc., have been recommended from time to time. Attacks sometimes may be stopped by the patient taking a long breath or a series of long breaths and fixing the chest in full inspiration, by the use of ice-cold douches to the chest, by irritation of the vagus nerve in the neck (pinching, faradization, etc.).

Bradycardia.—The term bradycardia is more or less loosely applied to abnormal infrequency of the pulse; supposedly the cardiac action is at the same time infrequent, but in reality this is not always so. Following Mackenzie, the cases of diminished frequency of the pulse may be

divided into four classes: (1) Those in which all the chambers of the heart participate in the slow action (true bradycardia); (2) those in which the slow pulse rate is due to a missed beat, the ventricle having contracted but the resulting pulse-wave being too feeble to reach the wrist (extrasystolic form of arrhythmia); (3) certain cases of nodal rhythm in which the auricle has ceased to beat, or beats synchronously with the ventricle (mild heart block); and (4) cases in which the stimulus is blocked between the auricle and the ventricle so that the auricle beats at its normal rhythm and the ventricle does not respond to the auricular systole, but pursues an independent and slow rhythm (complete heart block). The cases are due then to stimulation of the vagus (inhibition), or to depressed conductivity and contractility of the myocardium.

True bradycardia may be physiological or pathological. The physiological cases comprise certain normal individuals in whom the pulse is always abnormally slow—60 or less; the pulse may become slowed also during pregnancy, less than 60, even 50, 40, or less, and in starvation (but this perhaps verges on the pathological). The pathological cases comprise those due to: (1) Infections, such as typhoid fever, pneumonia, diphtheria, influenza, rheumatism, etc., in convalescence from which it is rather common; (2) intoxications, such as alcohol, coffee, tea, lead, tobacco, uremia, digitalis poisoning, etc.; (3) disease of the heart, especially the fatty heart, chronic myocarditis, aortic stenosis, etc.; (4) disorders of the lung, especially emphysema; (5) divers disorders of the gastro-intestinal tract, such as gastric carcinoma, jaundice, cholelithiasis (especially during colic), etc.; (6) nervous disorders, such as apoplexy, meningitis, brain tumor, injury of the medulla, vagus stimulation by tumors, coma, epilepsy, etc.; and (7) anemic, cachetic, and exhausting disorders of wide diversity. In true bradycardia the pulse rate is rarely less than 40. The condition is not serious; it may last for many years. The use of atropine, since it accelerates the cardiac activity in the vagus cases, may help to distinguish these from the myocardial cases.

DISEASES OF THE MYOCARDIUM.

In the introductory remarks on the pathological physiology of the cardiovascular system the prime importance of the myocardium has been emphasized, and later certain disorders to which the myocardium is subject under divers circumstances have been grouped together under the designation neuromuscular disorders. An attempt to separate diseases of the myocardium and to discuss them apart from other diseases of the heart, however, is a matter of extreme difficulty, if not impossibility. The relationships that the myocardium, in health and in disease, bear to the normal and the perverted activity of the heart are so intimate that one can scarcely be thought of independently of the other. Whatever the disorder of the heart under consideration, the major diagnostic, prognostic, and therapeutic considerations must be based upon the

condition of the myocardium. In chronic valvular disease, for instance, although the condition of the valves or orifices is of significance, the efficiency of the circulation, the comfort of the patient, and the duration of life depend upon the functional capacity of the myocardium under the new or changed conditions; this is true also in diseases of the pericardium, notably in chronic adhesive pericarditis, and likewise one may also say in the advanced anemic and cachetic stages of general disorders in which degenerative changes in the myocardium are prone to develop. The classification of diseases of the myocardium, therefore, is exceedingly intricate; no classification is entirely satisfactory. It is even difficult to draw a line between the primary and the secondary diseases of the myocardium. Hypertrophy and dilatation, for instance, are due to many factors, each more or less separate and distinct in itself and productive of varying grades of mischief; bacterial toxins, chemical poisons of widely diverse nature, and anemic conditions may engender degenerative changes in the myocardium that may be expressed in myocardial weakness or dilatation, the phenomena of which may dominate the clinical picture; the fibroid heart may be viewed as a disease of the myocardium or of the coronary arteries, to which in the great majority of cases it is directly due; and, finally, the nutrition of the heart under all conditions, its functional capacity, its ability to hypertrophy under the stress of unusual demands, its failure to adapt itself to new or changed conditions, etc., are due wholly to the condition of the coronary circulation—generally as well as locally. In the absence of a satisfactory classification, the subjoined arrangement of diseases of the myocardium, may be adopted tentatively; it comprises more or less well-defined clinical entities with associated pathological changes, which, however, gradually merge into other conditions.

THE WEAK OR INSUFFICIENT HEART.

(*Myocardial Weakness or Insufficiency; Congenital Weakness of the Myocardium; Acquired Weakness of the Myocardium; Cardiac Asthenia.*)

Etiology.—There is a more or less diversified group of disorders of the heart, of which the pathological lesions are inconspicuous, and of which the noteworthy clinical manifestation is myocardial weakness; the other clinical manifestations and the etiological factors may be quite dissimilar. These disorders, therefore, not only differ more or less among themselves, but they cannot be sharply separated from other disorders of the myocardium. Unquestionably some of these cases are congenital: the subject is born with a "weak heart;" other cases are acquired. In the congenital cases the weakness occasionally is more or less obviously represented in smallness in the size of the heart or in thinness of its walls; often the structure is apparently normal, but the organ is physiologically inadequate; in either event the heart is unable to meet the requirements of ordinary life. The acquired cases result

from damage to a heart originally normal. The important etiological factors are: (1) The infectious diseases. In these disorder of the heart may develop as described under the infectious febrile heart; usually the heart returns to its previously normal condition, but it may remain for some time or permanently weak or insufficient. (2) Intoxicants—such as alcohol, tobacco, coffee, tea, etc., especially when taken in excess. (3) Anemic and cachectic states. (4) Neurotic disturbances, exemplified in undue reaction to emotional and extraneous influences, or in general nervous incapacity with special involvement of the heart. In some cases the damage is doubtless not confined to the myocardium, but involves also the nervous mechanism; but the discoverable changes are inconspicuous.

Symptoms.—Many persons, subjects of congenitally weak hearts, conscious perhaps of other cardiac deficiency, or readily made aware thereof upon slight exertion, lead a quiet and retired life—well within the functional capacity of their hearts—and suffer no noteworthy ill consequences. Others, however, endeavoring to attain the average of human activities, suffer upon slight or moderate exertion, with precordial distress or oppression, palpitation of the heart, rapid heart action (120 or more per minute), with an unstable or irritable pulse, dyspnœa, general muscular weakness, etc. These symptoms may become of high grade and associated with more serious evidences of cardiac inadequacy should the patient lead an altogether ill-advised life, engage in athletic exercises, attempt feats of mountain climbing, etc. (consult the overstrained heart); or they may develop precipitously after some sudden severe exertion (consult acute dilatation of the heart).

In the acquired cases the symptoms, as a rule, are analogous. In the postinfectious cases the patient during quietude does not suffer, but more or less distress, palpitation, and dyspnœa ensue upon activity, and the patient is generally weak. In the alcoholic and tobacco cases the patient may be conscious of palpitation, fluttering, or irregularity of the heart even when quiet, and dyspnœa develops upon exertion. These symptoms are often associated with other evidences of intoxication, such as so-called indigestion, general nervousness, headache, and other mental symptoms. The disorder of the heart, of course, gradually passes over into well-recognized anatomical lesions (degenerative, chronic, fibrotic, etc.).

In the anemic and cachectic cases, in addition to the general manifestations of anemia, there is more or less precordial distress, cardiac palpitation, rapid heart action, weak apex beat, feeble pulse, dyspnea, syncopal attacks, etc.; these are referable in part to the anemia as such, but they are due also in part to the weakened and usually dilated myocardium the result of insufficient nourishment. The condition gradually merges into parenchymatous and fatty degeneration and chronic dilatation of the heart.

The neurotic cases are well exemplified in the more or less fleeting attacks that occur usually in neurotic women under most diverse circum-

stances, and are manifested by sensations of stoppage of the heart or precordial fluttering, pallor of the lips and face, and a general feeling of apprehension, "sinking," impending syncope, or paresthetic phenomena; the pulse is usually weak and somewhat accelerated (not the pulse of syncope —which does not occur). The attack lasts a variable time, and may be followed by considerable exhaustion. In other cases, especially following a long period of mental overwork, worry, etc., the heart, sometimes quite suddenly, becomes weak and may necessitate the patient's taking to bed; in aggravated cases any position other than the prone leads to syncope, weak heart action, and feeble pulse. The apex beat is weak, the first sound is short and valvular, the second sound is rarely accentuated; the action of the heart is often irregular and readily disturbed in rate and rhythm by various influences. There are no hysterical symptoms. Recovery may not ensue for several or many months.

Diagnosis.—The diagnosis of the acquired cases is not difficult as a rule. The congenital cases often pass unnoticed, until at least more or less dilatation has ensued, whereupon the condition is scarcely to be distinguished from dilatation arising from other causes. In the early stages the history of the patient, the absence of etiological factors, the development of symptoms upon the slightest exertion and their aggravation upon exertion well borne by the average individual, and the absence of noteworthy physical signs, aside from the rapidity and overaction of the heart and smallness and frequency of the pulse, should serve for the recognition of the disorder.

Prognosis.—In the congenital cases the outlook depends upon the mode of life of the patient, upon his living within the capabilities of his heart. The postinfectious cases usually eventuate in recovery, but this may be long delayed. The cases due to alcohol, tobacco, tea, coffee, etc., are likely to improve in the course of time, if the causative poison is discontinued and irreparable damage has not been done.

Treatment.—The treatment is largely hygienic. In the congenital cases the patient should be well nourished and should be given tonics to improve the general blood condition and nutrition, and he should indulge in carefully regulated exercise under competent supervision with a view to strengthening his myocardium; considerable improvement often follows. The treatment of the postinfectious cases is similar; iron is often a very useful remedy, but rest to the disabled heart may be required for a long time. Digitalis, strychnine, and similar drugs often do harm; they may be required, in the event of failing cardiac compensation, but rapid or unstable action of the heart of itself is not an indication for their use. The cases due to alcohol, tobacco, and the like usually improve with discontinuance of the causative factor, but the bromides, valerian, and aconite often render material service in quieting the overactive heart. The transient disorders that occur in neurotic women demand little else than stimulation and other measures useful in syncope; speaking generally, there is no danger in the attacks except that which lies in the habit of taking drams to cure or

prevent them (Allbutt). The more severe and prolonged neurotic cases are best treated by rest in bed, massage, hydrotherapy, nutritious food, strychnine, alcohol, iron, and arsenic. Later, benefit accrues from carefully regulated exercises.

THE OVERSTRAINED OR IRRITABLE HEART.

(Cardiac Strain; Muscular Strain of the Heart; The Soldier's Heart; The Athlete's Heart.)

Etiology.—The term overstrained heart is used as a convenient designation of the condition that results from excessive muscular effort. Probably, however, the term might be employed to comprise in addition all the conditions of the heart in which the myocardium, relatively or absolutely, is at a disadvantage (functionally insufficient) in relation to the intracardiac and arterial pressure, and in which in consequence overstrain ensues; these comprise cases of chronic valvular disease, the fibroid heart, diseases of the coronary arteries, arteriosclerosis, etc., all of which are discussed under appropriate headings. Although it may be difficult to estimate the importance of strain in an individual case, inasmuch as it is often bound up with other etiological factors of cardiovascular disease, such as syphilis, alcoholism, gout, plumbism, renal disease, etc., its prime importance is manifest when we compare the heart disorders of men with those of women of the same station in life; and when we compare the heart disorders of men engaged in laborious occupations, such as draymen, ironworkers, blacksmiths, etc., with those of men not engaged in laborious occupations, but equally but not more given to the vices of intemperance and sexual immorality, such as clerks, business and professional men. In estimating the etiological significance of strain, one must take into consideration all of the factors in the individual case; and one may no more cursorily and without investigation attribute to strain certain manifest diseased conditions in a patient than he may elect to overlook the possible etiological importance of a sudden and severe strain to which the patient himself may attribute the onset of his ailment. It is quite conceivable, indeed, it is quite true, that the healthy individual—the individual with his muscles, heart, bloodvessels, and lungs developed proportionately to the demands that he makes upon them—may endure a considerable amount of prolonged or sudden strain without obvious ill consequences. This we observe almost every day in athletes and like persons. But we also observe very frequently the disastrous results to the heart and the general economy of sudden severe strain and of prolonged strain ill-advisedly persisted in, not only in a certain proportion of those engaged in athletic contests, but also in those whose occupation necessitates considerable strain, in those poorly endowed by nature, who soon use up their reserve strength, and in those with hearts weakened by typhoid fever, influenza, and other infections, anemic states, etc.

Cardiac overstrain, in the restricted sense in which it is here employed, results: (1) From sudden violent muscular effort, such as may occur in lifting unusually heavy weights, in rowing races and similar contests, etc.; and (2) from repeated, long-continued, and ill-directed muscular exertion, such as may occur in college and other athletes, in all persons who engage unduly and inadvisedly in exercise, in soldiers subjected to forced and prolonged marches, etc. This overstrain of the heart, in view of the etiological factors, is largely a disorder of early adult life and of the male sex, but it is sometimes encountered in young women overzealous in the pursuit of athletics. It may develop in a heart previously normal, as well as in a heart damaged by myocardial or valvular disease or weakened by general causes, such as fever states, anemia, etc.

Pathology.—In sudden death from acute heart strain, all the chambers of the heart are dilated and filled with blood, and the lungs and the venous system are markedly engorged. In the chronic cases the heart shows more or less hypertrophy and dilatation, myocardial degeneration, perhaps mitral endocarditis, coronary arteriosclerosis, etc.; in a word, the more common concomitants of general and coronary arteriosclerosis.

The chief causative factors of cardiac overstrain are increased arterial pressure and increased and accelerated flow of venous blood to the heart, both of which are provoked by muscular exertion. The increased arterial pressure occasions an insufficient and incomplete emptying of the left ventricle, which in turn leads to a gradually increasing residuum of blood within, and dilatation of, the ventricle; at the same time the increased and accelerated flow of venous blood leads to a similar condition of the right ventricle. Dilatation of the right ventricle is the more common in the ordinary athletic contests, and can commonly be demonstrated by increased cardiac dulness to the right and a systolic murmur in the tricuspid region (tricuspid insufficiency; safety-valve action of the tricuspid valve). With cessation of the causative factor the heart, if previously healthy, reverts to its normal condition and no ill consequences ensue. Permanent damage, however, may follow the too frequent repetition of athletic contests or other muscular exertion, ill-directed athletic training, the non-adaptation of exercise to the individual—which is the essential etiological factor; this permanent damage occurs especially in young adults of poor physique, such as many college students, soldiers, etc. In the case of soldiers, to the other etiological factors one may add the weight of accoutrements, which embarrass respiration, the fatigue of prolonged marches, the abuse of tobacco and alcohol, venery, etc., all of which may gradually lead to what Da Costa so aptly described as the soldier's "irritable" heart.

Symptoms.—A person subjected to severe muscular exertion in excess of the capabilities of his heart, complains of a sense of fulness, or distress beneath the sternum and in the region of the heart, perhaps of actual pain that may radiate throughout the chest and into the arms; there is also palpitation of the heart, dyspnœa, general muscular weakness, vertigo, and sometimes even syncope. Observation discloses a

livid and anxious expression, with prominent eyes and dilated pupils, and excessive perspiration; fulness and pulsation of the veins of the neck; marked and widespread precordial pulsation; increased area of cardiac dulness especially to the right, with loud, clear, valvular heart sounds, a systolic murmur in the tricuspid area (relative tricuspid insufficiency; safety-valve action of the tricuspid valve), perhaps a systolic murmur elsewhere, and a weak pulse. These phenomena are to be interpreted as the manifestations of an acutely embarrassed heart. As a rule, especially in previously healthy subjects, they subside and leave no ill consequences; in rare instances, especially in those the subjects of previous heart disease, they may lead to an immediately fatal issue; in other cases, the acute phenomena subside, but the patient finds himself in the future more or less incapacitated. The original distressing symptoms and embarrassed cardiac action are again provoked in the athlete by contests, such as football, basket ball, running, rowing, bicycling, etc.; in some cases even moderate exercise serves to bring on the symptoms; whereas, especially in older subjects with perhaps some myocardial degeneration, even the slightest exertion (climbing stairs or a hill), perhaps even a full meal, or too much tobacco or alcohol, or constipation with intestinal distention, leads to more or less distress (precordial distress, dyspnœa, palpitation of the heart, irregular or quickened heart action, etc.); that is, the heart is unusually sensitive to extraneous influences well borne by a normal heart: it is said to be irritable. Comparative comfort or freedom from distress may be secured by the patient's leading a quiet life and avoiding all muscular strain. The physical signs may be entirely normal, or there may be signs of hypertrophy with some dilatation of the heart.

The more chronic cases, such as result from long continued excessive muscular exertion, present the common evidences of increased arterial pressure and hypertrophy of the heart, together with a marked tendency to the occurrence, upon exertion, of the aforementioned signs of cardiac embarrassment. The physical signs are those of hypertrophy and dilatation of the heart, of which the one or the other may predominate in the individual case.

Diagnosis.—In the acute cases the history and the mode of life of the individual are of great importance in determining the cause of the symptoms, especially in cases in which the physical signs are equivocal. The effect of exertion purposely undertaken under the observation of the physician may be of service in establishing a diagnosis. In the chronic cases the diagnosis depends upon a history of long continued overexertion and the physical signs of heightened blood pressure and dilated hypertrophy of the heart.

Prognosis.—In the acute cases the outlook depends entirely upon the future life of the individual, upon his avoiding the causes known to provoke cardiac distress. The prognosis in the chronic cases is that of hypertrophy and dilatation of the heart.

Treatment.—Overstrain of the heart should be prevented—by thorough physical examination of the heart of all intending athletes, soldiers, etc., by the avoiding of muscular exertion obviously too severe for the subject, and by proper training under supervision. By graduated exercises a weak but otherwise normal heart may be made stronger, and capable of sustaining greater efforts. Sudden severe overstraining of the heart often leads to more or less irreparable damage to the heart. The acute symptoms are best met by rest in the recumbent posture and diffusible stimulants, such as whiskey, brandy, aromatic spirit of ammonia, compound spirit of ether, etc.; in rare cases bleeding may be required. After recovery from these acute symptoms, rest for a long time is imperative. The patient must avoid all factors that in the least tend to irritate or accelerate the heart: not only exercise, but also alcohol, tobacco, tea, coffee, etc. After the lapse of several months graduated exercise, most carefully supervised, is often of service. The overacting, irritable heart may often be quieted by the use of the bromides, valerian, aconite, etc. Cardiac tonics, such as digitalis, strychnine, etc., are of little service, except in the more chronic cases with dilatation, the treatment of which is that of failing cardiac compensation in general.

THE FATTY HEART.

Etiology.—The term fatty heart is applied to two conditions, fatty degeneration and fatty infiltration. Although they may occur together, in well-marked cases, they represent two quite distinct diseases. Fatty degeneration usually follows and is associated with parenchymatous degeneration, and is due, therefore, to like causes; but it occurs also in many forms of hypertrophy, in old age, etc. Fatty infiltration or overgrowth is an excess of the normal subpericardial fat, and is a part of general obesity.

Pathology.—Usually the fatty deposit is most marked along the course of the larger coronary vessels and in the auriculoventricular furrows; but it may entirely envelop the heart in a thick casing and may infiltrate between the muscular fibers even to the endocardium. In some regions the muscular fibers may be entirely replaced by the fatty tissue. Aside from the fatty deposit, the heart may be quite normal, but it is usually somewhat enlarged, hypertrophied in consequence of excessive food and drink, and dilated in consequence of muscular weakness. In addition to the fatty deposit there is usually some fatty degeneration and simple and brown atrophy of the muscular fibers; the coronary arteries often reveal some sclerosis, and there are consecutive areas of fibrosis of the myocardium.

It is difficult in many cases to determine to what degree the symptoms are due to the fatty deposit. It is not improbable that a large encasement of fat more or less seriously compromises the functional activity of the heart, but there are many cases of well-marked fatty infiltration of the

heart that present no symptoms until the advent of dilatation, and many fatty hearts with symptoms reveal in addition coronary sclerosis and fibroid myocardium—which may well be answerable for the symptoms. Perhaps in many cases the fatty deposit is secondary to primary atrophy of the muscular fibers.

Symptoms.—The majority of obese subjects sooner or later complain of dyspnœa, slight cyanosis, and palpitation of the heart on exertion (these are often due, in part at least, to the obesity itself and interference with the action of the muscles of respiration); others have periodical attacks of so-called asthma (perhaps induced by overeating or overdrinking), or of bronchitis; still others complain of vertigo, pseudo-apoplectic attacks, with Cheyne-Stokes breathing, anginoid pains, or angina pectoris. Examination of the heart (rendered difficult by the excessive panniculus adiposus) reveals increase in the area of cardiac dulness and weak or muffled cardiac sounds; and the pulse is small and weak. In the course of time, unless properly and efficiently treated, the symptoms of failing compensation (dyspnœa, passive congestion of the viscera, and œdema), with a murmur of relative mitral, and perhaps tricuspid, insufficiency, may develop; or the patient may die suddenly in an anginoid attack.

Diagnosis.—The diagnosis, which rests upon cardiac enfeeblement in an obese subject, is always precarious; the symptoms doubtless are often due less to the fat about the heart than to associated lesions.

Prognosis.—The prognosis virtually is that of obesity plus the associated cardiac conditions.

Treatment.—The treatment is that of obesity, associated with appropriate measures to invigorate the action of the heart. The so-called Oertel treatment (regulated diet, largely protein, limitation of the fluid intake, and graduated exercises up hill) here finds an especial field of utility.

THE INFECTIOUS FEBRILE HEART.

(*Myocardial Degeneration; Parenchymatous and Fatty Degeneration of the Heart; Acute Myocarditis.*)

The infectious febrile heart is a somewhat inapt and perhaps ill-chosen term designed to describe the condition of the heart in the acute infectious processes; the lesions are chiefly degenerative, but to these in some cases inflammatory changes are added.

Etiology.—The etiological factors are the toxins of the acute infections generally, but notably, acute rheumatic fever, typhoid fever, pneumococcic and other pyococcic infections (septicopyemias), influenza, diphtheria, etc. These toxins perhaps act upon the nervous mechanism, stimulating the accelerator nerves, since fever of itself is accompanied by an accelerated heart-rate. The chief influence of the toxins, however, is exerted on the myocardium and the vasomotor system.

Pathology.—The changes vary considerably in different cases. In some they are slight and apparently inconsequential, transitory in nature, and leave no permanent ill consequences. In others there is a varying reaction of the myocardium to the toxin; whereas in still others there may be also endocarditis or pericarditis, or what may be properly spoken of as carditis or pancarditis. Endocarditis and pericarditis are described elsewhere, but it is perhaps wise to point out here that most of the symptoms and signs of these disorders, such as precordial distress, dyspnœa, cardiac and pulse irregularities, changes in the size of the cardiac dulness, as well as some murmurs, etc., are of myocardial origin.

At the necropsy, after a fatality in one of the aforementioned acute infections, the myocardium, to the unaided eye, is usually pale and opaque, and flabby or friable; occasionally it is streaked with yellowish lines, especially that of the left ventricle and the papillary muscles. The heart may be more or less dilated; the cavities may be filled with laminated and anemic clots; but sometimes in sudden death they are quite empty (vasomotor collapse). Microscopically the muscle fibers are swollen and opaque, the seat of more or less advanced albuminous, granular, or parenchymatous degeneration (cloudy swelling). The lesions, if unchecked, proceed to fatty degeneration (the yellowish streaks in the muscle of the ventricles or the papillary muscles); or there may be associated hyaline or amyloid degeneration. The nerves may reveal degenerative changes. The interstitial tissue often reveals little if any change. In some cases, especially of severe infections, or associated with endocarditis or pericarditis, there is, in addition to the myocardial degeneration, more or less vascular dilatation and cellular exudation and proliferation (acute interstitial myocarditis). The lesions may subside or give rise to a form of chronic myocarditis. In septicopyemia there may be embolism of the small coronary vessels, with scattered foci of suppuration containing the causative microörganisms (suppurative myocarditis). In favorable cases the small abscesses become inspissated and a cicatrix ultimately forms, leading to a form of chronic localized myocarditis, with perhaps the later development of an aneurysm of the heart; in other cases the abscess may increase in size and ultimately rupture into the pericardium or a cavity of the heart.

Symptoms.—From the nature of the etiological factors cardiac symptoms are often in abeyance, being masked by those of the primary disorder. In many of the infections there may be no complaint whatever while the patient is at rest in bed; dyspnœa and palpitation of the heart, however, not infrequently follow even slight exertions, and excitability of the heart is shown in attacks of rapid, irregular, and intermittent action upon slight "starts," emotional and other disturbances, etc. (toxic depression of conductivity and the dropping out of ventricular systoles). In other cases the pulse is persistently rapid. In the majority of infectious or toxic cases the symptoms develop as the patient, convalescing, begins to substitute the activity of returning health for the prone and quiet posture of disease. The first symptom may be a sudden and fatal

attack of heart or vasomotor failure, such as not infrequently occurs during convalescence from diphtheria. In other cases less severe attacks of heart failure occur; syncope attends the upright posture or the getting out of bed; in other cases the heart becomes rapid, and is subject to periods of irregularity and intermittency on exertion or excitement. But in a notable series of cases the patient presents general evidence of cardiac inadequacy (depression of tonicity); he complains of muscular lassitude and of cold hands and feet and nose; while generally pallid, his toes and finger tips and nose and ears may be bluish (capillary stasis); he is mentally torpid and does not respond to mental or other stimuli; the urine is of low specific gravity, reduced in amount, and may contain a slight trace of albumin; and there may be vomiting. In many of these cases, to the cardiac inadequacy vasomotor failure is added; this may readily be determined by a rapid pulse and low blood pressure. Systolic pressures of 100 mm. Hg. are not uncommon; less than 80 mm. may be found in severe infections, and is of serious moment. In unusual cases, instead of rapidity there may be unusual slowness of the heart action, proceeding even to heart block. Influenza sometimes attacks the heart with special severity. During the attack there may be marked prostration, with dyspnœa, cardiac palpitation, syncope, and very feeble heart sounds; after the attack has subsided dyspnœa and tachycardia often persist, and may be associated with precordial pain and distress or anginoid pains on exertion.

Examination reveals the heart action rapid, irregular, or intermittent; usually but not always there is some dilatation; the apex beat is weak and diffuse; the first sound at the apex is muffled and prolonged, or weak, short, and valvular; or it may be replaced by a variable blowing systolic murmur (relative mitral insufficiency from stretching of the mitral ring, or from irregular or incoördinated action of the papillary muscles); the pulmonary second sound is usually accentuated; and the blood pressure, as a rule, is low (often less than 100 mm. Hg.), but fluctuates much from time to time and is unusually responsive to divers sorts of extraneous influences. Added sounds (extrasystoles) may be audible. The heart may also be observed to dilate under exertion or excitement.

Diagnosis.—The diagnosis should be apparent from the etiological factors and the obvious evidences of weak, irritable, or unstable cardiac action, especially a rapid, irregular, and feeble, or a very feeble, slow pulse, capillary stasis, weak apex beat, weak and valvular first sound of the heart, evidences of dilatation, variable apex murmur, and low blood pressure.

Prognosis.—As a rule, most cases eventuate in recovery with the cure of the initiating disorder; but, as in the influenzal and other cases, this may be longer delayed. Occasionally the heart remains for a long time or permanently susceptible, and dyspnœa and palpitation develop on slight provocation.

Treatment. Attempts should be made to minimize the damage to the heart by the use of antitoxin in diphtheria; by hydrotherapeutic measures

to reduce the fever, especially in cases of long continued or high fever; and by free elimination in all toxic conditions. Absolute rest in bed is the most important element of the treatment; it should be prolonged especially in diphtheria, scarlatina, severe pneumonia, influenza, etc., and the getting up and about should be gradual and controlled by the effect on the heart and the pulse. Especial attention should be paid to the diet, which should be wholesome, highly nutritious, and readily digestible. Iron, quinine, the bitter tonics, and alcohol are the most suitable remedies; these may be augmented by cold affusions and other simple hydrotherapeutic measures, which are of the utmost value in restoring tone to the cardiac muscle and the vasomotor system. Cardiac tonics, such as digitalis, strophanthus, strychnine (except in very small doses), are contraindicated, in the early stages at least. In protracted, slowly progressing cases, small doses of digitalis often prove serviceable. In these cases also massage and graduated exercises are of value.

Attacks of acute failure of the heart are to be met with diffusible stimulants, such as aromatic spirit of ammonia, compound spirit of ether, brandy, whiskey, hot coffee, etc., by the mouth; or whiskey, caffeine, camphor in oil, strophantin, adrenalin chloride, etc., hypodermicly, with adrenalin chloride and physiological saline solution by the bowel, hypodermically, or intravenously. The acute failure with marked dilatation of the heart is sometimes promptly relieved by venesection.

THE FIBROID HEART.

(*Chronic Interstitial Myocarditis; Fibroid Degeneration of the Myocardium; Sclerosis of the Coronary Arteries.*)

Etiology.—In the great majority of cases the fibroid heart is a result of sclerosis of the coronary arteries, which in turn may be a more or less independent disorder, but usually is merely part of general arteriosclerosis; the causes of the one, therefore, are the causes of the other, notably alcoholism, syphilis, habitual and excessive muscular work; but, as in arteriosclerosis generally, excessive eating, gout, prolonged and excessive mental efforts, lead poisoning, etc., also are of significance. The disorder, therefore, is more common in the male sex and after the fortieth year; and in some cases it seems to be predisposed to by hereditary influences. Occasionally chronic myocarditis results from or is a sequel of acute myocarditis, representing the healing and cicatrization of the acute lesions; or it is associated with endocarditis and pericarditis, representing an extension into the myocardium of the lesions of either of these two disorders; or it is intimately bound up with the conditions of hypertrophy and dilatation, and of the so-called fatty heart. Finally, the disorder may be merely an expression of advancing age.

Pathology.—The lesions may be diffused or circumscribed. The circumscribed lesions implicate especially the left ventricle, more particularly the apex, and less frequently the septum, the right ventricle,

the papillary muscles, and the auricles. They consist of firm, whitish streaks or patches, of variable size, interspersed between the muscular fibers. Usually these areas can be demonstrated to be directly related to sclerotic changes in the finer branches of the coronary arteries. In consequence of lessening of the lumen of these small vessels (obliterating endarteritis) the portion of myocardium thereby supplied with blood becomes imperfectly nourished, gradually undergoes degenerative and atrophic changes, and is ultimately replaced by connective tissue. Occasionally the interruption in the arterial supply is sudden (thrombosis or embolism), and if the patient does not die immediately, an area of anemic or hemorrhagic infarction ensues and is usually replaced by a scar. Associated with the fibrosis there is more or less hypertrophy and dilatation, varying in grade in different parts of the heart. Since a heart ill supplied with blood cannot hypertrophy, the hypertrophy must be due to extracardiac factors, notably an antecedent valvular disease, general arteriosclerosis, chronic nephritis, idiopathic hypertrophy, or localized hypertrophy of the non-involved portion of the myocardium, etc.; the dilatation is the result of the gradual yielding of a weakened myocardium to the intracardiac pressure. Sometimes the yielding is limited to, or marked at, a small area (scar of a healed infarct), and a so-called cardiac aneurysm results; exceptionally this or the initial area of infarction may rupture and give rise to sudden death. In those cases in which there is extensive fatty change, the sclerosis has usually involved the larger branches of the coronary arteries rather than the smaller twigs, and leads to a general interference with the nutrition of the myocardium.

Symptoms.—The symptoms are extremely variable. In some cases there are no noteworthy symptoms whatever, more or less extensive myocardial fibrosis being found after death from other causes. In these cases, of course, the demands made upon the myocardium were still within the limit of its capabilities. Symptoms when they develop are those of embarrassed or insufficient heart action, developing, as a rule, gradually in a man past middle life. Sometimes the symptoms are precipitated by an intercurrent disorder, such as pneumonia or other infection, or by some intoxication, such as an alcoholic debauch or an attack of acute gout, or by some unusual exertion or excitement precipitating dilatation of the heart. In the cases beginning thus, more or less suddenly, the symptoms are those of more or less well-marked cardiac decompensation. In the cases of more gradual development the patient begins to notice general sallowness of the integument, weakness, and languor; he is readily tired, and less capable than formerly of mental or physical exertion: the first induces fatigue and mental depression, and the second dyspnœa, palpitation of the heart, precordial distress, and perhaps syncopal attacks (cerebral anemia). With increasing dilatation of the heart, the phenomena of failing compensation (cyanosis, œdema, passive congestion of the viscera, etc.) develop. Another series of cases is characterized by anginoid pains or attacks

of true angina pectoris. In another series of cases the phenomena of the Stokes-Adams syndrome develop; or a person apparently in good health dies suddenly (coronary thrombosis and anemic infarcts, fatty degeneration of the heart, or extensive fibrosis); or the symptoms may be merely those of advancing age.

Examination of the heart reveals variations in accordance with associated conditions, arteriosclerosis, chronic nephritis, valvular disease, chronic pericarditis, emphysema, etc. Usually there is more or less hypertrophy with dilatation. The apex beat is displaced downward and outward in accordance with the predominating hypertrophy and dilatation, and it is feeble; often, indeed, it is entirely absent. The first sound of the heart varies: it may be feeble or muffled, suggesting irregularity of the muscular contractions; or it may be loud and valvular (disproportionately loud as compared with the feeble apex beat) suggesting more or less complete loss of the muscular element; or, and this is quite significant, there may be a distinct gallop rhythm. This may be of the presystolic type, in which the new third sound immediately precedes the normal first sound of the heart, and is due to a strongly acting hypertrophied auricle, or, if heard in weak and rapidly acting hearts, is due to a delay in conductivity and sufficient separation in time of the auricular and the ventricular contractions to enable one to differentiate the separate sounds produced by both; or the gallop rhythm may be of the protodiastolic type, in which the new third sound occurs in the early part of diastole immediately following the normal second sound, or it may be mesodiastolic, in which the new sound occurs in the middle of diastole; in both of these the new sound is ventricular in origin and is perhaps due to loss of tonicity of the ventricular muscle and its sudden distention by an ample and rapid volume of blood. The second sound may be reduplicated, but this is less frequent than in valvular disease. There are commonly no murmurs until the disease is fairly well advanced, when a systolic murmur of relative mitral insufficiency and perhaps also of tricuspid insufficiency, may ensue, the consequence of incoordination of the papillary muscle or dilatation of the arterioventricular rings. The cardiac action and the pulse are often irregular in rhythm and force; the pulse is usually small and feeble; it may be rapid, but it is often characteristically slow, 60, 50, or less to the minute, and not uncommonly exhibits extrasystoles (pulsus bigeminus).

Diagnosis.—The diagnosis is often difficult and sometimes impossible, but special significance attaches to the manifestation of a failing or embarrassed heart in a middle-aged person without the etiological factors and concomitant signs of valvular disease; and especially to a slow, irregular pulse, attacks of anginoid pains or syncope, and evidences of sclerosis of the palpable arteries.

Prognosis.—The prognosis is always grave, since restitution to the normal is impossible; but with care and the adoption of a suitable mode of life the patient may live many years.

Treatment.—In the cases of gradual onset and slow progress, as well as in the heart of fatty overgrowth, much can often be done to arrest the progress of the disorder by regulation of the mode of life, especially as regards eating and drinking, and by properly regulated rest and exercise. The so-called Oertel cure (regulated diet, largely protein, limitation of the fluid intake, and graduated exercises up hill) and the Schott or Nauheim treatment (effervescing baths and resisted exercises) are often very advantageous. Cases developed upon a syphilitic basis may perhaps be benefited by the use of the iodides, which also may be given in any case of arteriosclerosis; benefit sometimes attends its prolonged use. Cases of obvious cardiac insufficiency, manifested by dyspnœa, cyanosis, œdema, and rapid and irregular pulse, etc., are to be treated as is the decompensation of valvular disease. The cases with slow pulse and no signs of dilatation are to be treated with diffusible and other stimulants, and with morphine (in the event of restlessness, nervousness, anxiety, etc.), and nitroglycerin (in the event of high blood pressure); digitalis is contra-indicated.

HYPERTROPHY AND DILATATION OF THE HEART.

(*Enlargement of the Heart.*)

Enlargement of the heart may be due to hypertrophy, to dilatation, or to both. By simple hypertrophy is meant a simple increase in the thickness of the muscular walls without increase in the size (or capacity) of the heart cavities; by eccentric hypertrophy is meant increase in the thickness of the muscular walls with increase in the capacity of the cavities. Increase in the thickness of the walls with diminution in the size of the cavities probably never occurs (formerly so-called eccentric hypertrophy).

Etiology.—Hypertrophy and dilatation, although often the obtrusive evidence of disorder of the heart, are, in the great majority of cases, the expression of the activity of more or less remote causative factors. In most cases the hypertrophy and dilatation, which are usually combined, are readily referred to a manifest valvular defect; but occurring independently of valvular disease, the hypertrophy or dilatation is frequently overlooked, or, being detected, is set down as a primary disorder, when in reality it is a result or part and parcel of other diseased conditions.

In general, hypertrophy or dilatation is an expression of the functional capacity of, and of the demands made upon, the heart; it represents the relationship between the power possessed by the heart and the work that it is called upon to perform. In health there is a balance between these, and, to the astonishment of all who think of it, the heart that never ceases its activity remains normal. When, upon occasion, the heart is called upon temporarily to perform some unusual work, its reserve force is such that it is usually enabled to do it without damage to itself. Such, however, may be the inherent weakness of the heart, or the dis-

proportionately great amount of work that it is called upon to perform, that it succumbs (cardiac overstrain), and acute dilatation results. On the other hand, the increased amount of work may be moderate in amount but frequently repeated, in which circumstance the heart fortifies itself by hypertrophy—in many respects a conservative process. In this manner the heart is enabled to perform the increased amount of work for some time—until its nutrition suffers or the work becomes too great, whereupon dilatation and the usual evidences of embarrassed circulation, of failing cardiac compensation, result. Thus, dilatation, both acute and chronic, not infrequently occurs without associated hypertrophy; but whether hypertrophy ever occurs independently of dilatation may well be doubted—most if not all cases of so-called concentric hypertrophy reported being examples of postmortem contraction of the heart.

Hypertrophy and dilatation may affect the heart as a whole, or may be confined to one side or to one of the four great divisions of the organ. Usually there is associated disease of both sides, and not uncommonly of all four divisions. The hypertrophy and dilatation of valvular disease is discussed under the appropriate headings; occurring independently of valvular disease, there are the following clinical varieties: (1) Acute dilatation; (2) chronic dilatation; and (3) hypertrophy, or, as it is sometimes called, dilated hypertrophy.

Acute Dilatation.—Acute dilatation is a common event in the course of many of the acute infective diseases, and not infrequently leads to death (see the infectious febrile heart). Acute dilatation may be observed also in certain chronic diseased states, such as are attended by grave secondary anemia and degenerative alterations of the myocardium. In some of these cases the heart, gradually failing, may suddenly dilate, although chronic dilatation is the more common event. Acute dilatation results also from sudden violent exertions—from the attempting of work altogether disproportionate to the natural and reserve force of the heart. Thus, we find it in draymen and ironworkers who suddenly exert all their muscular power in the effort to lift heavy weights; in athletes, oarsmen, runners, football players, etc., after participation in some unduly strenuous contest; in mountain climbers, especially in those who attempt difficult ascents to which they are unaccustomed, etc. It is a feature also of what is known as angiospastic dilatation of the heart.

Chronic Dilatation.—Chronic dilatation, occurring independently of valvular disease, is common in states of general malnutrition attended by grave secondary anemia. It attends fatty degeneration of the heart muscle, myocarditis, fibroid heart, and pericardial adhesions. It is a terminal event in hypertrophy from whatever cause arisen, unless the subject dies of an intercurrent affection. It results from gradual weakness (loss of tonicity) of the heart muscle, which becomes less and less able to perform its allotted work.

Hypertrophy.—Hypertrophy of the heart, occurring independently of valvular disease, may be due to a variety of causes. The hypertrophy

that effects especially the right ventricle is due to increased pressure in the pulmonary circulation, and is found most commonly associated with emphysema, fibroid lung, chronic bronchitis, chronic pleural adhesions with contraction, etc. It is really a part of these diseases—a conservative process on the part of the heart, a condition that arises in response to increased demands upon the heart. It may persist for years, enabling the heart to do the increased work well, and the patient may die of an entirely independent affection. In many cases, however, dilatation and its attendant phenomena ultimately result.

Hypertrophy of the left ventricle, occurring independently of valvular disease, results from increased pressure in the systemic circulation, and from increased activity of the heart. Among the rarer causes are congenital hypoplasia of the aorta, such as is sometimes observed in chlorosis and other conditions; narrowing of the aorta, usually congenital and occurring in the neighborhood of the ductus Botalli; aneurysm of the aorta, usually, however, associated with general arteriosclerosis or aortic valve disease; pressure on and obstruction of the aorta, such as may be observed in intrathoracic tumor and indurative mediastino-pericarditis, etc. Practically, however, in the majority of cases, hypertrophy of the left ventricle partakes of one of three varieties: (1) That due to arteriosclerosis; (2) that due to renal disease; and (3) that, for want of a better name, known as idiopathic hypertrophy of the heart. In some cases it is difficult to refer the hypertrophy to one or the other of these classes, inasmuch as two or three may be concerned in the causation of the hypertrophy in the individual case. Thus, arteriosclerosis and renal disease are frequently associated, and the subject of one or both of these is frequently exposed to the causative factors of the so-called idiopathic hypertrophy.

The hypertrophy of *arteriosclerosis* is common in advanced life—when to a considerable extent it is a conservative process. But it occurs frequently enough in relatively early life, when it is bound up etiologically with syphilis, alcoholism, plumbism, rheumatism, gout, etc. In the early stages the hypertrophied and contracted arterioles oppose a barrier to the onward movement of the blood, and high tension results. To overcome these, the heart, reacting to the stimulus of increased work, hypertrophies. Later the arteries become rigid and more or less incapable of contracting. Thus, an important adjunct to the onward movement of the blood—the activity of the arteries themselves—is lost, and the heart must compensate by hypertrophy. Ultimately the entire arterial system may become extremely rigid and permanently distended, whereupon the heart must furnish almost if not quite all the power for the movement of the blood, and this is possible only through marked hypertrophy. At the same time, the coronary arteries participate in the process, the nutrition of the heart does not keep pace with its increase in size, and chronic dilatation ensues.

The hypertrophy of the heart of *renal disease* occurs in all types but not in all cases of nephritis; it is most common and most marked in

chronic diffuse indurative (or interstitial) nephritis, in which it is properly looked upon as part and parcel of widespread disease of the cardiovascular and renal systems. In the majority of cases the disorder doubtless commences in the kidneys, but it speedily implicates the heart and the blood vessels; in some cases the lesions begin simultaneously in the kidneys and in the cardiovascular apparatus. The primary factor is always increased blood pressure, which is discussed in detail under nephritis

Idiopathic Hypertrophy of the Heart.—In a certain proportion of cases of cardiac hypertrophy (usually associated with some dilatation) none of the ordinary causes can be determined upon. There is neither valvular disease nor disease of the coronary or systemic arteries; no disorder of the myocardium as such, no pulmonary emphysema or other disease of the lung, no chronic nephritis, no arteriosclerosis, and yet there is cardiac hypertrophy. Eliminating the other and rarer causative factors previously mentioned, these cases—for want of a better term—are known as cases of idiopathic hypertrophy of the heart.

Some at least of these cases are due to nervous causes, to persistent overactivity of the heart induced by undue irritability or overstimulation of the nervous mechanism. This is sometimes seen in nervous persons, in those susceptible and unduly exposed to psychic disturbances; it is found also in some cases of exophthalmic goitre; and is sometimes due to the misuse of tea, coffee, tobacco, etc.

Another class of cases is undoubtedly due to excessive eating and drinking. There can be little doubt that excessive eating temporarily increases the specific gravity of the blood, and excessive drinking the total volume of the blood; these, together with the stimulating effect of certain metabolic products, result in increasing temporarily the blood pressure and the frequency and quickness of the heart's action. The frequent repetition of this calls for more work on the part of the heart, and when its reserve force has been exhausted hypertrophy ensues. This is seen well exemplified in the great hypertrophy of the heart in beer drinkers (the Munich beer-heart), and in the obesity of indulgence, especially in men over forty years of age. Alcohol in these cases is probably a contributing factor, but it is probably less answerable for the hypertrophy (which, as already mentioned, is to a certain extent a conservative process) than it is for arterial alterations and subsequent degenerative changes in the heart.

Finally, one of the most interesting causes of cardiac hypertrophy is excessive muscular strain (which has been discussed under the over-strained heart).

Pathology.—The conspicuous alterations are increase in the size of the heart, increase in the thickness of the walls, and change in the shape. The normal heart weighs from 250 grams (woman) to 300 grams (man); in hypertrophy with dilatation the weight is often twice or thrice this, sometimes more. In health the left ventricle measures 8 to 14 mm. in thickness, the right 4 to 7 mm., the left auricle 2 to 3 mm., and the

right auricle 1 to 2 mm. When diseased, the left ventricle may measure 20 to 25 mm., the right ventricle 8 to 15 mm., the left auricle 3 to 4 mm., the right auricle 2 to 3 mm. The papillary muscles share in the hypertrophy. When there is marked dilatation, thinning of the walls is often more apparent than real, being relative only. The cavities of the heart are greatly increased in capacity, being doubled, or even tripled; such may be the distention that relative insufficiency of otherwise normal valves ensues. The heart is variously altered in shape, depending upon the portion of the heart especially affected. In general, the apex becomes broader and blunter; this is especially the case in hypertrophy of the right ventricle, in which the heart as a whole becomes globular, and the entire apex may be made up of the thickened right ventricle; in hypertrophy of the left ventricle, although the apex is broader than normally, the heart still preserves its conical shape. When dilatation supervenes the heart becomes generally rounded. In the early stages of hypertrophy the heart muscle is firm to the touch, and dark red brown in color; in increasing dilatation, however, it loses its firmness, and becomes flaccid, and in high degrees of dilatation it is commonly a misshapen, toneless mass of tissue at the necropsy. Microscopically there is an increase in the number and size of the muscular fibers; in late stages fatty degeneration of the myocordium and fibroid alterations ensue.

Symptoms.—Hypertrophy of the heart, since it is a conservative process on the part of nature, frequently exists for a considerable time without occasioning symptoms; indeed, by its presence it obviates certain symptoms that otherwise would develop on account of the diseased condition which it endeavors to correct. Sometimes, however, there are unpleasant sensations in the region of the heart, evidences of overactivity of the heart, precordial distress, rarely amounting to pain, subjective appreciation of the heart's action, etc. These are especially noticeable after exertion or excitement, when they may be accompanied by slight dyspnœa, vertigo, etc. Sooner or later, however, there are symptoms. These, in large part, are not to be distinguished from those of failing cardiac compensation occurring in the course of myocarditis or chronic endocarditis. The early symptoms in such cases, however, differ. Thus, in athletes and others whose hypertrophy is due to excessive muscular exercise, and who rather suddenly cease this muscular activity, there may be the subjective manifestations of overactivity of the heart, precordial distress, dyspnœa, cough, hemoptysis, tinnitus aurium, vertigo, flushings, disturbed vision, etc. (consult the overstrained heart). In other cases more or less constant and persistent dyspnœa precedes the final breakdown.

The symptoms of acute dilatation are those already mentioned under acute cardiac overstrain and under the infectious febrile heart. The symptoms of chronic dilatation are those of decompensation, described under chronic valvular disease of the heart—precordial distress and pain, cough, dyspnœa, cyanosis, œdema, congestion of the viscera, etc.

Physical Signs.—The physical signs vary somewhat, depending upon the portion of the heart affected.

Left Ventricle.—Inspection and palpitation reveal bulging of the precordium, which in young subjects may cause marked asymmetry of the chest, widening of the intercostal spaces, increase in the force of the apex beat, and a widely diffused heart beat. The apex beat may be as low as the sixth, seventh, or eighth intercostal space, and 2 to 5 cm. outside the mid-clavicular nipple line (13 to 18 cm. from the median line). The apex beat is peculiarly deliberate and heaving in character, increased in strength, and about 2 fingers' breadths in width. In some cases of well-marked hypertrophy a double apex beat may be felt, the second being attributed to a rebound of the heart transmitted from the aortic valve. Percussion reveals an increase in the area of dulness, which is more ovoid than in health, the long axis of the ovoid being directed in the long axis of the heart. The dull area (deep or relative dulness) may extend upward to the third rib, rarely to the second interspace, to the left 2 to 5 cm. beyond the mid-clavicular line, while to the right, in uncomplicated hypertrophy of the left ventricle, the increase of dulness is relatively slight—not more than 2 cm. ($\frac{3}{4}$ inch) beyond the right edge of the sternum. On auscultation, if the valves are still competent, the heart sounds are normal, or the first sound is dull, somewhat louder, and more prolonged than normally (due to increase of the muscular element). The aortic second sound is usually accentuated, sometimes reduplicated or split. The pulse is regular, of good volume, increased in strength and in tension.

Right Ventricle.—Inspection and palpation reveal bulging of the precordium, especially at the xyphoid cartilage and adjacent right cartilages, with some widening of the intercostal spaces. The heart-beat is diffuse—the strongest beat not infrequently being at the lower part of the sternum. The apex beat also is diffuse, wider than normally—2 to 3 fingers' breadths in width—and extends to the left beyond the mid-clavicular line; it is rarely dislocated downward, and when so, not below the sixth intercostal space. Percussion reveals great increase in the deep cardiac dulness transversely, to the left slightly, and to the right considerably—2 to 5 cm. ($\frac{3}{4}$ to 2 inches) beyond the right border of the sternum. The area is irregularly ovoid—transversely ovoid. On auscultation the heart sounds may be normal; the first sound usually is slightly dull and prolonged, and the second sound is accentuated or reduplicated in the pulmonary area. The pulse is usually regular, and increased in volume and tension.

Hypertrophy of the auricles does not occur in the absence of associated disease of the ventricles or of valvular disease, and is scarcely to be diagnosticated in the absence of such associated disease. Hypertrophy of the left auricle commonly causes enlargement anteroposteriorly, since that the chamber of the heart, as a rule, does not come in contact with the chest wall; upon occasion, however, there may be some increased dulness in the third or the second left intercostal space,

together with a visible and palpable presystolic (auricular systolic) pulsation. Hypertrophy of the right auricle gives rise to dulness to the right of the sternum and a visible and palpable presystolic (auricular systolic) pulsation in the third and fourth right intercostal spaces, occasionally also in the third left intercostal space.

With the onset of dilatation the apex beat grows weaker, more diffuse, and irregular; often, although a diffuse wave may be seen, none can be felt (or scarcely felt); the dulness is increased, especially to the right, the heart sounds lose their muscular element and become sharp, higher-pitched, valvular, and later quite feeble and indistinct. Murmurs of relative mitral and tricuspid insufficiency frequently develop; gallop rhythm may supervene; autochthonous pulsations appear in the jugular veins and the liver; the pulse becomes small, quick, and irregular; and other evidences of failing cardiac compensation develop. At times death occurs suddenly, and without premonition.

Prognosis.—The prognosis depends upon the etiological factors and the associated conditions. Hypertrophy itself is a conservative process; so long as it remains efficient the outlook is good. From the nature of the process, however, the final prognosis is bad, since ultimately the nutrition does not keep pace with the enlargement of the heart, and degenerative processes and finally dilatation ensue. The prognosis in dilatation depends upon its cause and degree.

Treatment.—The treatment of hypertrophy and dilatation is so intimately bound up with the causal factors that it can scarcely be discussed apart from them. In general, an overactive hypertrophied heart is much benefited by rest, the avoidance of tobacco, coffee, tea, alcohol, etc., and the use of sedatives, such as the bromides, valerian, aconite, etc. The treatment of dilatation is that of decompensation in valvular disease.

ANEURYSM OF THE HEART.

Aneurysm may involve the myocardium or the valves. Valvular aneurysm may result from acute valvular endocarditis, which leads to softening and weakness of a leaflet, and permits of its distention in response to the influence of the blood pressure. This is most common at the aortic orifice, where the aneurysm usually forms an ovoid or spheroid projection into the left ventricle; rarely a similar condition occurs at the mitral valve. Rupture may occur and give rise to valvular insufficiency. Otherwise the condition cannot be recognized during life.

Myocardial aneurysm may follow an area of localized weakening; this is usually due to fibroid myocarditis, a scar of a healed infarct, a gumma, mural endocarditis, etc. The common seat is the left ventricle near the apex. The aneurysm is usually saccular, rarely a rather diffuse dilatation. There are no characteristic symptoms. Occasionally there may be a localized pulsating prominence near the region of the

apex beat, and an irregular area of dulness. The condition may be revealed by a Röntgen-ray examination. The *prognosis* is bad, rupture ensuing eventually. *Treatment* is unavailing.

RUPTURE OF THE HEART.

Rupture of the heart is a rare event that may occur in cases of degenerated or softened myocardium. This occurs especially in aneurysm of the heart, in the fibroid heart, in the fatty heart, in softening (myomalacia cordis) due to obstruction (embolism or thrombosis) of the coronary artery, in suppurative myocarditis, gummas, ulceration endocarditis, etc. It occurs most commonly in men over sixty years of age, and is usually due to some exertion. The rupture may occur anywhere, but is most common in the wall of the left ventricle near the septum. Occasionally, also a valve leaflet may be ruptured and lead to valvular insufficiency; usually it results from severe exertion or traumatism to the chest, or from ulcerative endocarditis. The *symptoms* of rupture of the heart are rarely unequivocal. Usually there is sudden death without premonitory symptoms. Occasionally there is sudden, perhaps agonizing, pain in the precordium, a sensation as of something giving way in the chest, dyspnœa, anxiety, collapse, and death from the pressure of the ensuing hemopericardium upon the heart; death may be delayed for twelve hours or more, rarely, even for days. The *diagnosis* is rarely made; it is suggested by the aforementioned symptoms and the physical signs of fluid in the pericardium. The *prognosis* is hopeless and *treatment* ineffectual.

TUMORS AND PARASITES OF THE HEART.

Tumors of the heart are rare; but lipoma, fibroma, myoma, sarcoma, and carcinoma have been observed. Sarcoma and carcinoma are usually secondary, and may be single or multiple. There are no significant symptoms; decompensation may ensue ultimately.

Parasites of the heart also are rare. The most common is Echinococcus granulosa. Cysticercus cellulosæ, Trichinella spiralis, Streptothrix actinomyces, etc., have also been observed.

DISEASE OF THE CORONARY ARTERIES.

Disease of the coronary arteries can scarcely be separated from disorders to which it gives rise or with which it is associated. The integrity of the myocardium, of course, depends upon an efficient coronary circulation—which may become disordered in toto or locally, slowly or suddenly. The chief consequences are: (1) Infarction and softening. This is due to occlusion of a larger or smaller twig (end artery), and is

most common in the area of distribution of the anterior coronary artery. The absence of such infarction in some cases of arterial occlusion is attributable to abnormal anastomoses or to an efficient circulation through the vessels of Thebesius. The softened infarcted area is gradually replaced by scar tissue, which later may be the seat of an aneurysm or of rupture of the heart. (2) The fibroid heart (which see). (3) Angina pectoris (which see). (4) Sudden death. A not uncommon cause of sudden death is occlusion of one coronary artery or of one of its main branches. The occlusion is usually due to thrombosis of a vessel somewhat sclerosed and more or less narrowed; the circulation has been perhaps for some time precarious, but, the final interruption of the circulation being rather sudden, death may ensue immediately.

DISEASES OF THE ENDOCARDIUM.

ACUTE ENDOCARDITIS.

Etiology.—Acute endocarditis is rarely a primary disorder; usually it is secondary to infectious processes elsewhere in the body. It occurs most frequently in association with rheumatic fever, the likelihood of its development increasing with recurrence of attacks of rheumatism; 75 per cent. or more of endocarditic subjects are or have been rheumatic, and others give a history of such related disorders as tonsillitis (the portal of entry of the infectious agent), chorea, neuromuscular (growing) pains (especially significant in children), erythema nodosum, etc. Acute endocarditis occurs also in association with scarlatina, pneumonia, pyococcic, gonococcic, and typhoid infections, dysentery, influenza, and occasionally also with diphtheria, tuberculosis, measles, etc. The local lesions in the heart may be provoked by the primary infective agent, but frequently they are set up by secondary pyococcic invaders. The disorder is most common in adolescents and young adults, corresponding with the period of greatest prevalence of the aforementioned infections. It is predisposed to by diabetes, chronic nephritis, gout, syphilis, carcinoma, and chronic anemic and debilitating disorders in general, and in consequence it is not uncommonly a terminal event in older subjects. It is often engrafted on an old or chronic endocarditis.

Pathology.—Different types or forms of the disease may be distinguished—rheumatic, staphylococcic, streptococcic, pneumococcic, gonococcic, typhoid, influenzal, etc.; simple (or benign) and ulcerative (or malignant); verrucose and ulcerative; and valvular and mural. Except etiologically, no sharp line of demarcation can be drawn between these types.

The lesions are most common in the left side of the heart, the relative frequency being as follows: The mitral valve in 50 per cent. or more of

the cases when affected alone, in 80 per cent. when associated with lesions elsewhere; the aortic valve in 15 per cent. when affected alone, in 40 per cent. when associated with lesions elsewhere; the tricuspid valve; the pulmonary valve; the wall of the left auricle and ventricle; and the wall of the right auricle and ventricle. Endocarditis developing during intra-uterine life is more common in the right side of the heart, which fact is attributed to the greater strain and stress to which the right and the left side of the heart respectively is subjected in intra-uterine and extra-uterine life; there are, however, other more or less ill-understood factors, such as anomalies of development, etc.

In the simplest form, the lesions consist of small, grayish, translucent vegetations or excrescences attached to the line of closure of the valves, that is, to the surface opposed to the blood current, the auricular surface of the mitral valve, and the ventricular surface of the aortic valve. Often the vegetations are extremely small, 1 to 2 mm. in diameter, bead-like; they may be larger, and have a fissured or wart-like surface; sometimes they are attached by a pedicle, that is, polypoid. They are most commonly situated along the line of closure of the valves, which appears to be related to the fact that the regions most exposed to strain and traumatism are most likely to be less resistant, and that the infection is probably effected through the medium of the blood circulating through the cavities of the heart, rather than that circulating in the coronary vessels, although this is still a debated point. The vegetations developing on the valves may spread to the chordæ tendineæ, the wall of the cavities, etc. They consist for the most part, of fibrin, leukocytes, and blood platelets (coagulable elements of the blood), superimposed upon an area of desquamating and proliferating endothelial cells. Occasionally there is a surrounding area of reactive congestion, but often this is entirely absent. The causative microörganisms may be found in the vegetations, but they are not infrequently absent when sought—which suggests that, having initiated the lesions, the microörganisms may die out, or, as is likely, that toxins suffice to set up the lesions. Insufficiency of the valve is a common concomitant, being the result of malposition of the leaflets dependent upon the vegetations or upon a consecutive deformity of the leaflets; but when the vegetations are small the insufficiency is often due less to the vegetations per se than to myocardial disorder and interference with the normal action of the valve leaflets and papillary muscles.

The subsequent course of events varies: (1) In the course of time the proliferating endothelial cells and fibroblasts may invade the vegetation, and by a process akin to organization gradually lead to its replacement, so that ultimately the lesion is represented by a scar. In rare cases, in which the lesions have been minor in grade, restitution to the normal may perhaps ensue; but this is doubtful, and very unlikely in a given case. The scar usually contracts and leads to thickening, induration, and distortion of the valve leaflets—chronic valvular disease. (2) The lesions may progress to what is often spoken of as ulcerative or malignant endocarditis. This, merely an aggravation of the more benign lesion,

usually begins on the valves, but it is likely to spread to the chordæ tendineæ and the mural endocardium, and while it is most frequent in the left side of the heart, it is not uncommon also in the right side. It is characterized not only by the most exuberant vegetations, but also by more or less extensive necrosis and ulceration of the underlying tissues; so that to the previously existing valvular insufficiency, stenosis of the orifice (from blockage by vegetations) is not infrequently added. The ulceration may be superficial, involving perhaps only the endocardium; but usually it is more extensive, and not uncommonly leads to aneurysmal dilatation and perforation of a valve leaflet, destruction of the chordæ tendineæ or papillary muscles, perhaps considerable necrosis, suppuration, even perforation of the cardiac septa or walls. The lesions may also spread to the aorta and the pulmonary vessels. The vegetations are likely to become disintegrated, and being detached in part or in whole and swept into the circulation, cause embolism and infarction in different organs, especially the spleen, kidneys, brain, intestine, etc. (or in the lung in involvement of the right side of the heart). Multiple abscesses (so-called arterial pyemia) ensue, if the emboli are infected. The lesions in the heart thus are often relatively insignificant, being overshadowed by those of the primary disorder and the metastatic phenomena. Often the cardiac lesions are widespread, involving the endocardium, the myocardium, and the pericardium, so that the term pancarditis is most appropriate.

Symptoms.—The milder grades of acute endocarditis frequently run their course entirely unsuspected; of themselves they occasion no noteworthy or unequivocal symptoms, and frequently are disclosed only by the necropsy. Since the disease is especially common in rheumatic fever, the likelihood of its occurrence should be borne in mind; its development should be suspected in the event of precordial distress or oppression, palpitation, dyspnœa on slight exertion, rapid and irregular cardiac action, and persistence or sudden increase of fever without adequate articular or other signs to explain it. The more marked grades of the disorder exhibit gradual progression in symptomatology to that of frank septicopyemia—irregular chills, fever (remittent or high and continuous), sweats (often profuse), rapid loss of flesh and strength, progressive anemia, polynuclear leukocytosis, enlargement of the spleen, diarrhœa, pyococcic or other bacteremia, etc. These phenomena are not essentially different, whether they occur in septicopyemia attended or unattended with endocarditis; but they are especially significant if there be no adequate cause to explain them, or if they occur in a subject of old valvular disease.

In most cases the diagnosis depends upon the occurrence of embolic phenomena and the results of a physical examination of the heart. Of embolic phenomena the most important and suggestive occur in the skin—petechiæ; in the kidneys—renal pain, albumin, blood, and tube casts in the urine; in the spleen—splenic pain, palpable and audible frictions; in the lungs—pain, hemoptysis, perhaps localized dulness,

bronchial breathing, and fine rales; in the cerebrum—hemiplegia, monoplegia, aphasia, blindness, convulsions; in the eye—retinal hemorrhages and optic neuritis, as well as petechial hemorrhages in the palpebral conjunctiva; in the long bones—local pain and tenderness; in the joints—local pain, swelling and tenderness; painful ephemeral erythematous nodules in the fingers, toes, hands, and feet; and in the extremities—gangrene from vascular obstruction. The joint symptoms are often fugitive and much resemble the articular manifestations of rheumatism; they are doubtless toxic in nature, as are also some of the skin manifestations, such as certain roseolas, erythemas, urticarias, etc.

The extreme variability in the symptomatology has led to a classification of the disorder into several types: (1) The cardiac type, in which cardiac distress and obvious valvular lesion with attendant failure of cardiac compensation constitute the noteworthy features; (2) the septic, or pyemic type, which cannot be distinguished from septicopyemia, of which in reality it is a manifestation; (3) the typhoid type, resembling typhoid fever in the toxemia, asthenia, and prostration; and (4) the cerebral type, resembling meningitis. These terms, however, are largely misnomers, since the disease necessarily is always cardiac and toxemic or septicopyemic, and the irregularity and multiplicity of the lesions and symptoms do not really permit of such classification.

The physical signs are extremely uncertain. In some cases there may be no discernible deviation from the normal, especially in some of the streptococcic, gonococcic, and pneumococcic forms. The cardiac action is often persistently excited (without obvious adequate cause), and sometimes peculiarly unstable, attacks of tachycardia (120 to 150 heart beats per minute) often developing suddenly and ceasing quite as suddenly (without apparent cause); this is of some diagnostic importance. In some cases the first sound at the apex becomes impure or muffled, and finally replaced by a murmur, and other signs of mitral insufficiency develop; in other rarer cases the signs of aortic insufficiency or other valvular defect ensue. Extreme variability in the character of the murmur from day to day (perhaps related to changes in the vegetations, or due to incoördinated action of the papillary muscles) is quite significant, but is not always present.

The clinical course of the disease is quite variable, but permits of grouping of the cases: (1) Mild cases, which are common in rheumatic fever, and occur also in typhoid fever and other infections and in debilitated conditions, perhaps as a terminal infection. The clinical phenomena are slight or altogether absent, but if the patient survives, the lesions usually lead to chronic valvular disease. (2) Severe cases, which are especially common in pyococcic infections (puerperal infection, wound infection, pneumonia, gonorrhœa, scarlatina, etc.), although they may occur also in milder infections, rheumatic fever, etc. The clinical phenomena may be more or less obtrusive, and the lesions may subside leaving a valve or valves more or less damaged; or a fatal result may ensue

within several weeks; or a chronic septic process may be set up and continue for months. (3) Recurring or relapsing cases, which may be interpreted as relighting of an obsolete infection or reinfection of a damaged valve—a form of chronic endocarditis.

Diagnosis.—The diagnosis of the simple or mild cases is difficult, and is often a matter merely of conjecture. Increase of the cardiac dulness, an apical systolic murmur, and an accentuated pulmonic second sound are not uncommonly due to toxic degeneration of the myocardium provoked by divers infections (rheumatic fever, etc.). The gradual development of the murmur is suggestive of endocarditis; but since anemia and degeneration of the myocardium usually supervene at about the time endocarditis may develop, the diagnosis cannot always be made, and often must await the passage of time; persistence of the physical signs after convalescence from the infection, of course, is conclusive evidence of a valvular defect. The development of a diastolic murmur during the infection is of itself almost conclusive of aortic endocarditis. The diagnosis of the more severe cases may be based upon the symptoms of septicopyemia, embolic phenomena, and the physical signs referable to the heart already mentioned. In obscure cases blood cultures may afford material diagnostic assistance.

Typhoid fever may be excluded by the absence of the prodromes, especially the persistent headache and nose-bleed, roseola, tympanites, delirium, dry tongue, Gruber-Widal reaction, and typhoid bacilli from the feces, urine, roseola, and blood, and by the presence of a source of infection, irregular and repeated rigors, intermittent fever, profuse perspiration, rapid and unstable pulse, undue dyspnœa, the early occurrence of marked prostration, the rapid development of anemia, leukocytosis, embolism, and the detecting of the infecting microörganism in the blood. The spleen is said to become less enlarged, although more tender, in endocarditis (and sepsis) than in typhoid fever. It is well also to bear in mind that the typhoid bacillus may set up the endocarditis, either primarily or by infecting an old lesion. In acute tuberculosis, as contrasted with acute endocarditis, variations in the temperature and pulse rate, and excessive perspirations occur with more regularity, wasting is more rapid, a local focus of tuberculous disease may be detected in some part of the body—in the lungs (tubercle bacilli in the expectoration), in the pleura, in the abdomen, in the brain (tubercle bacilli in the cerebrospinal fluid obtained by lumbar puncture), etc.; physical examination of the heart reveals no signs of valvular disease (except in the rare cases in which there is an associated valvular lesion); and embolism does not occur. The detecting of choroidal tubercles in the one case, and of retinal hemorrhages in the other, is of the greatest diagnostic value. Malaria may be excluded by the irregularity of the attacks of chills, fever, and sweats, polynuclear leukocytosis, absence of malarial plasmodia from the blood, and the administration of quinine.

Prognosis.—The prognosis varies with the severity of the lesions—which has already been referred to. The seriousness of the lesions,

however, sometimes cannot be judged from the clinical manifestations, so that for a time at least the prognosis as regards life may be doubtful; that as regards the heart is always bad, since chronic valvular disease almost certainly results. Rarely even severe so-called malignant endocarditis may eventuate in recovery, with more or less permanent damage to the heart. Usually, however, it ends in death, or the acute symptoms, fever, etc., subside within two or three months.

Treatment.—The treatment resolves itself largely into that of the primary infection—whether rheumatic or non-rheumatic. It is perhaps possible in some cases of rheumatism, to prevent the development of endocarditis—by prompt and efficient treatment of the rheumatism. The patient should be absolutely at rest in bed, and should be brought promptly under the influence of the salicyl preparations—large doses to the point of mild intoxication. This not only relieves the rheumatic manifestations, but, counteracting the poison it should tend to prevent the occurrence of endocarditis; there is some evidence that it does. Some observers believe that the addition of the alkalies (in twice the dose of the salicyl preparation) is of much value. Caton insists on the importance also of small repeated blisters to the precordium, and he as well as others extol the advantage of prolonged rest in bed—six weeks to three months.

The treatment of developed endocarditis also is largely that of the primary disorder—in rheumatic cases that just mentioned; in other cases, that of the initiating pyococic, gonococcic, pneumococcic infection, etc. Should it be possible to cultivate the provoking microörganism from the blood, a vaccine may be prepared and used with hope of good result. The serums used heretofore have not been especially successful. Colloidal silver also is scarcely to be recommended. Indeed, in severe cases, it is doubtful if any measures, aside from general supportive, can achieve much good. In the event of palpitation or cardiac distress an ice-bag should be applied to the precordium, or aconite may be used in small doses.

CHRONIC ENDOCARDITIS.

Etiology.—Acute endocarditis, if it does not lead to death, usually subsides within two or three months, and leaves the cardiac valves permanently damaged (chronic valvular disease), although rarely the lesions may be limited to the mural endocardium. In a few cases, however, the endocarditic phenomena are protracted—for many months; and in many cases, having become more or less quiescent, they are relighted (recurring or relapsing endocarditis). The etiological factors in these chronic cases are the bacterial agents that set up acute endocarditis; but the later attacks in the recurring cases are not always provoked by the bacterial cause of the primary attack; the damaged valves are especially vulnerable to the attack of any pathogenic bacterium.

Pathology.—The lesions vary much in different cases. In most cases (relapsing form) they consist of the various lesions more particularly described under valvular disease, to which the lesions characteristic of acute endocarditis are added; in some cases to the deformity of the valve, more or less exuberant, sometimes rather firm, vegetations are added; in other cases there are more or less superficial or deep ulcerations.

Symptoms.—The symptoms in general are like those of acute endocarditis, but protracted or recurrent. In one series of cases (chronic recurrent or relapsing endocarditis), aside from the symptoms due to the associated valvular disease, there are recurring attacks of chills, fever, and sweats, and leukocytosis (mild septicopyemia), with intervening periods of good health. During the attack the cardiac symptoms may be in abeyance, or those previously present may become aggravated, and the embolic phenomena of acute endocarditis may supervene. In another series of cases the symptoms are continuous from the beginning; they do not differ essentially from those of acute endocarditis, although perhaps they are less severe; but rarely chills, fever, sweats, and leukocytosis may last for months. In certain rare and protracted cases persistent fever (100° to 102°) is the characteristic and dominant manifestation; chills and sweats are much less common than in the acuter cases, but they may occur from time to time, and are then usually associated with an added increment of fever. Occasionally, after the lapse of months, the lesions may subside and the patient become apyretic, but recurrence of the symptoms is likely. Rarely there is a slight cough and the patient may emaciate (suggesting tuberculosis). There are usually the physical signs of mitral or aortic valve disease, although occasionally for long periods or throughout the disease there may be no murmur whatever. Embolic phenomena occur, but they are less common than in the acuter forms of the disease.

Diagnosis.—The basis of a diagnosis is the same as that of acute endocarditis; but special importance attaches to the knowledge of an old valvular disorder, increase in the size of the heart, changes in the character of the cardiac murmurs, the development of new murmurs, embolic phenomena, recurring attacks of fever, etc. In cases that present little else than persistent fever it may be quite impossible for some time to eliminate typhoid fever or tuberculosis. A blood culture may reveal the nature of the infection. Malaria should be readily excluded.

Prognosis.—The prognosis is bad. Rarely cases in which the symptoms continue unabated from the beginning may last a year or more. The relapsing cases may continue for several years, the duration being determined by the periods of apyrexia, the permanent damage done to the cardiac valves, and the ensuing changes in the myocardium.

Treatment.—The treatment is that of acute endocarditis plus that of the associated valvular disorder.

CHRONIC VALVULAR DISEASE OF THE HEART.

Etiology.—Two types of chronic valvular disease of the heart may be distinguished: (1) The chronic inflammatory, and (2) the arteriosclerotic or atheromatous; but a sharp line of distinction cannot always be drawn between them, and the final lesions in both are much alike. The first or chronic inflammatory type most frequently involves the mitral valve, is most common in young subjects and in women, and usually results from acute endocarditis; the causes of the one, therefore, are the causes of the other, the chronic lesions representing the results of healing of the acute process. In a minority of the cases, the lesions may be chronic from the beginning, initiated perhaps by the toxins of attenuated microörganisms or by certain ill-understood autotoxins. The second, arteriosclerotic or atheromatous type, most frequently involves the aortic valve, is most common in male adults, and occurs with increasing frequency in both sexes as the subjects advance in years. The especially important etiological factors are alcoholism, syphilis, and excessive and long-continued muscular strain, but it is due also to gout, plumbism, chronic nephritis, diabetes, and the divers other factors related causally to arteriosclerosis and high arterial tension. In some cases, especially of mitral valve disease, no etiological factor can be determined with certainty. A few cases, especially of aortic valve disease, are apparently traumatic in origin, developing in consequence of sudden undue muscular strain, severe contusions or compression of the chest, etc., which lead directly to rupture of a valvular leaflet. Heredity seems to play a role in some cases. Lesions of the right heart are usually congenital. Acute lesions are not infrequently superadded to the chronic: this may be a frank acute, more or less severe (or malignant) endocarditis, due usually to the pyococci; or it may be the relighting of a more or less quiescent or obsolete infection (recurring or relapsing endocarditis).

Pathology.—Among 1058 cases collected by Parrot, the mitral valve was involved in 621 (58.7 per cent.), the aortic in 380 (36 per cent.), the tricuspid in 46 (4.3 per cent.), and the pulmonary in 11 (1 per cent.); the left side of the heart in 1001 (94.7 per cent.), the right side of the heart in 57 (5.3 per cent.). In minor or slight cases the lesions consist of opacity, or of slight, perhaps nodular, thickening and induration near the free margin of the valve leaflets, just within the margin in mitral and tricuspid disease, and about the corpora aurantii in aortic disease; these may be interpreted as the results of so-called organization of the acute vegetations, or as the analogue of the early changes of arteriosclerosis (which may be associated). The body of the valve leaflets may reveal grayish-white opacities or yellowish areas of fatty degeneration. The newly formed fibrous tissue, of which the opacities and thickenings are made up, gradually undergoes contraction and leads to varying degrees of curling or puckering of the edge of the leaflets, or of stiffness, induration, shortening, and deformity of the leaflets as a whole. On account of

the consequent lack of resiliency the leaflets do not approximate properly, or on account of shortening, they do not approximate at all, and the condition known as insufficiency of the valve ensues. In the case of the auriculoventricular (mitral) valve this is often due to or augmented by involvement in the sclerotic and contracting process of the chordæ tendineæ and tips of the papillary muscles. In the course of time the adjacent edges of the leaflets often become adherent, in consequence of which their movement is still further restricted, and not receding as they should normally, they oppose a barrier to the onward movement of the blood—so-called stenosis or obstruction. Calcium salts frequently become deposited in the sclerotic and necrotic tissues, and add to the already existing rigidity of the leaflets. In advanced cases the normal tissues of the valves may be entirely replaced by a firm, fibrous, resistant calcareous mass capable of scarcely any movement, so that the valves are insufficient and the orifice stenosed. The lesions sometimes spread from the valves to the mural endocardium; rarely the mural endocardium alone is involved, a larger or smaller indurated, cicatricial, and perhaps calcareous patch being present. Superadded to the chronic, acute lesions are sometimes found. Hypertrophy and dilatation of the heart, varying grades of myocarditis, and coronary sclerosis are almost constant concomitants.

Pathological Physiology.—The heart and bloodvessels are so adjusted to the actual and potential work required of them, that in health and under the stress of unusual temporary functional demands, the required work is performed without discomfort to the patient. Sudden increased demands, such, for instance, as are developed during strenuous exercise, lifting unusually heavy weights, etc., are met with increased rapidity of the heart action; often slight dilatation of the heart ensues, but being well within the limits of the so-called reserve power of the heart, no serious consequences ensue; with the accomplishment of the work the dilatation subsides and the heart's action returns to the normal. Should the demands be more continuous, although perhaps less onerous, the heart responds, as does the musculature throughout the body, by hypertrophy, and the persisting dilatation that otherwise would ensue is counteracted, the equilibrium of the circulation is maintained: the lesion is said to be compensated. In valvular disease, in consequence either of orificial obstruction to the onward flow of the blood or of insufficiency of valves permitting regurgitation of the blood, the primary dilatation and consecutive hypertrophy involve first that chamber of the heart immediately back of the valve or orifice affected—the auricle in mitral and tricuspid valve disease, and the ventricle in aortic or pulmonary valve disease. Hypertrophy may not develop when the myocardium has become enfeebled in consequence of long-continued fever, general debilitating conditions, such as marked anemia, diabetes, carcinoma, amyloidosis, fibroid degeneration, etc. The hypertrophy when it does occur is sufficient to maintain the equilibrium of the circulation for a longer or shorter period, but it is not permanently efficient; to the hypertrophy

dilatation is gradually added, and as the dilatation, in consequence of insufficient nourishment and consecutive degeneration of the hypertrophied myocardium, assumes the ascendency, the period of compensation is supplanted by a period of unstable compensation, and finally by a period of failure or lack of compensation (decompensation). During the period of compensation the valvular disorder is disclosed only by its physical signs or by a lessened ability on the part of the heart to respond to unusual demands without discomfort to the subject (dyspnœa, cyanosis, palpitation of the heart, etc.); this the consequence of lessened reserve power. Gradually, as the myocardium becomes enfeebled, it becomes less able to withstand the vicissitudes of ordinary life, and it dilates. The ensuing loss of compensation may develop suddenly and rapidly lead to death; usually it is of gradual onset and progressive; it may be temporary or permanent. For a time the heart is manifestly adequate to meet the demands of quiet life, but more or less distress (dyspnœa, cough, cyanosis, palpitation of the heart, precordial pain, vertigo, faintness, slight and transient œdema about the ankles, etc.) ensues upon exertion, excitement, a full meal, etc.; but the symptoms subside upon the restoration of quiet: the compensation is unstable. Sooner or later the heart becomes unable to maintain the equilibrium of the circulation even when the body is at rest; the aforementioned symptoms become more or less permanent: the compensation is lost. This condition may eventuate in death; but in many cases the symptoms gradually subside temporarily, and are followed by recurring relapses and partial or more or less complete recoveries.

The first evidences of this failure of compensation are manifest in that part of the cardiovascular system immediately back of the chamber of the heart primarily involved: in valvular disease of the left side of the heart, therefore, in the lungs; but in most cases the lungs have been for some time engorged, so that speedily the congestive and other disturbances become manifest in the general venous system throughout the body. At the same time, failure of compensation becomes apparent in front of the lesion in a lessened and irregular blood supply. The evidences of lack of compensation, then, are disclosed not so much by the physical signs referable to the heart, although these are important, as by the evidences of general venous and capillary stasis and a small, weak, frequent, and irregular pulse.

The ordinary mechanism of valvular insufficiency and stenosis is that already described. Under other circumstances, however, the valvular orifices dilate, and normal leaflets become, therefore, incompetent to close the enlarged orifice—a condition spoken of as relative insufficiency, and very common at the mitral and tricuspid orifices. It doubtless occurs, but is very rare, at the aortic and pulmonary orifices. Relative obstruction (or stenosis) is occasionally produced by exuberant vegetations, ball-valve thrombus, aneurysmal dilatation of a leaflet, etc.; and a condition sometimes spoken of as relative stenosis ensues when the cavities

become much dilated while the orificial ring remains normal or is relatively only slightly enlarged.

Mitral Insufficiency.—Mitral insufficiency is the commonest lesion, occurring alone in about 30 per cent. or more of the cases; mitral stenosis occurs in about 16 per cent., and both lesions in about 12 per cent.

Etiology.—Mitral insufficiency may result from one of three factors: (1) Acute or chronic endocarditis. This is the common cause of the disorder in young persons; usually it is a sequel of rheumatism, "growing pains," chorea, scarlatina, and other infections. The mitral leaflets may be thickened, indurated, curled up at the edge, or everted, and contracted or puckered (the contraction and thickening may also involve the chordæ tendineæ), so that the leaflets cannot be approximated; or more or less of one or both leaflets and the adjacent chordæ tendineæ may be eroded or destroyed; rarely a leaflet may be perforated. In some cases the adjacent margins of the leaflets become adherent, and as the newly formed connective tissue contracts, the leaflets are drawn together, so that to the insufficiency obstruction is added. Acute lesions are not uncommon (recurring or relapsing endocarditis). (2) Slowly progressing sclerosis, analogous to arteriosclerosis. This is a less common form; it is due to the ordinary causes of arteriosclerosis, and is found in adults and elderly persons. The leaflets and the chordæ tendineæ are usually thickened and contracted, and the seat of more or less well-marked atheroma with calcification. Associated obstruction is not uncommon (12 per cent. of the cases). Minor grades of atheroma of the mitral valve are common in persons upward of sixty years of age, but usually do not lead to valvular defects. (3) Dilatation of the mitral ring. This, so-called relative or muscular insufficiency, is perhaps the commonest form. It is found in a wide variety of disorders in which the myocardium becomes reduced in tone and permits of such dilatation of the mitral ring, and usually also of the left ventricle, that the normal mitral leaflets are unable to close the orifice: anemic conditions, infectious fevers, hypertrophy and dilatation of the heart, aortic valve disease, arteriosclerosis, chronic nephritis, chronic myocarditis, nervous disorders, such as exophthalmic goitre, etc.

Pathological Physiology.—In consequence of incompetency of the mitral valve, a portion of the ventricular blood, varying in amount with the degree of incompetency, is returned to the left auricle during the ventricular systole, that is, at a time when the auricle in diastole is receiving blood normally from the lungs. The immediate result of this is an abnormal rise of the intra-auricular pressure toward the end of auricular diastole and consequent dilatation of the left auricle. The tension and stretching to which the auricular musculature is thus subjected stimulate it to vigorous contractions, in an effort to expel the augmented amount of blood, and the constant recurrence of the stimulus ultimately induces more or less hypertrophy. The regurgitating stream impedes the flow of blood from the pulmonary veins into the left auricle and leads immediately to stasis in the pulmonary circulation—

since the pulmonary veins are unprovided with valves, and the supposed constriction of the musculature at the insertion of the pulmonary veins into the auricle cannot long remain active or oppose an effective barrier to back pressure into the lungs. The increased pulmonary pressure acts as an obstruction to the movement of blood from the right heart and stimulates the right ventricle to more vigorous contractions and soon leads to hypertrophy—which is the effective factor in maintaining compensation. The increased volume of blood injected into the left ventricle with each auricular contraction leads to a corresponding dilatation of the left ventricle; this, in turn, is followed by more vigorous contractions in an effort to discharge the augmented volume of blood, so that ultimately some hypertrophy of the left ventricle ensues, but in uncomplicated mitral insufficiency this is rarely marked. So long as the hypertrophy of the right ventricle remains efficient, the mitral lesion is compensated and the patient may not suffer any distress. Sooner or later, however, the musculature weakens and dilates, either in consequence of myocardial degeneration or of increase in the mitral lesion; and relative insufficiency of the tricuspid valve ensues. The back pressure then is communicated to the right auricle and the liver and the general venous system. In mitral insufficiency the lungs are always engorged, and ultimately the condition of cyanotic induration develops (congestion and dilatation of the bloodvessels, hemorrhages, pigmentation, and fibrosis); the larger pulmonary vessels may exhibit atheromatous changes. When the back pressure finally involves the general venous circulation, passive congestion ensues, and in the course of time leads to œdema of the subcutaneous tissues, chronic catarrh of mucous surfaces, cyanotic induration of solid organs, transudates in the serous cavities, etc.

Symptoms.—Well-compensated mitral insufficiency may be disclosed only by a physical examination of the heart; there may be no subjective symptoms whatever, although usually some little embarassment of respiration and hurried action of the heart ensue upon sudden or severe exertion, such as hill climbing, ascending stairs, or strenuous exercise. In the majority of cases, however, the patient, although not seriously embarassed during ordinary activities, is made aware of his cardiac deficiency upon any unusual exertion; others may lead a quiet life with comfort, but even minor activities induce cardiac distress—which exerts its chief effects on the pulmonary circulation, and becomes manifest in palpitation of the heart, precordial distress, dyspnœa, and slight cyanosis of the cheeks, lips, ears, and finger tips. The congestion of the lungs disposes to attacks of bronchitis, which are readily induced by seasonal and climatic changes, and to attacks of hemoptysis (less frequent than in mitral stenosis). In long-standing cases that develop in youth, the finger tips tend to become clubbed.

Sooner or later the heart begins to fail, more or less complete, temporary or permanent, loss of compensation ensues; the passive congestion of the lungs becomes more marked, and to this is added the evidences of general venous and capillary stasis. The patient complains of palpitation, and

the heart action becomes rapid, irregular, and weak. More or less cyanosis develops. Dyspnœa, cough, and perhaps expectoration are expressions of the increased pulmonary engorgement; the expectoration is usually mucous, sometimes watery or serous, occasionally streaked with blood; usually it contains pigmented alveolar epithelial cells (so-called heart disease cells). The congestion of the liver (enlarged and tender) may lead to slight jaundice or lend a subicteric hue to the conjunctivæ and the skin. Dropsical effusions supervene, at first in the subcutaneous tissues in the dependent portions of the body. For a time they are transitory, developing during the day when the patient is up and about, and disappearing when he is in bed; but they soon become more or less permanent, although fluctuating; they may involve the general subcutaneous tissues (anasarca), and may occur also in the serous cavities of the body. The right pleura is most frequently involved—four or five times as frequently as the left, and when there is bilateral involvement, the collection of fluid is usually greater in the right side. This was attributed by Steele and Stengel to pressure on the azygos veins by the dilated right auricle; more recently Fetterolf and Landis, stating their belief that the fluid comes, not from the parietal, but from the visceral pleura, attribute the pressure factor to compression and partial occlusion of the pulmonary veins by dilated portions of the heart—dilatation of the right auricle in right-sided transudations, and dilatation of the left auricular appendix and of the left ventricle, and perhaps also retrodisplacement of the ventricular septum, in left-sided transudations. The urine becomes reduced in amount and contains a trace of albumin and often erythrocytes and tube casts. Insomnia, often distressing, and perhaps slight delirium at night may develop. Usually there are recurring attacks of such failure of compensation; under appropriate treatment the patient recovers and may again enjoy good health. Eventually, however, compensation becomes permanently lost and the patient dies of gradually increasing cardiac weakness and dilatation; but sudden death may ensue.

Physical Signs.—Depending upon the youth of the subject when the disorder developed and the degree of hypertrophy of the right ventricle, there is more or less bulging of the precordium; the cardiac impulse is forcible and widespread, often especially conspicuous toward the sternum or in the epigastrium; the apex beat (the palpable beat farthest outward and downward) is displaced outward transversely (usually not downward in uncomplicated mitral valve disease); it is of increased breadth and not infrequently somewhat heaving in character. A systolic thrill may be palpable at the apex, and sometimes the forcible closure of the pulmonary valve may be palpable in the pulmonary area as a diastolic shock. The deep (and the superficial) cardiac dulness is increased transversely—somewhat to the left, but mostly to the right, forming a transverse oval that may extend one to two inches or more to the right of the sternum. Sometimes there is an increase of the dulness upward and outward from the left edge of the sternum—attributable to

enlargement of the left auricle (but enlargement of the left auricle is usually predominantly fore and aft, and usually causes little if any increase in dulness upward). At the apex a systolic murmur is usually audible—soft and blowing in character and low pitched, but it may be harsh and high pitched or musical; it may partly or completely replace the first sound; it may be audible only in the early part of systole (when it often progressively diminishes in intensity), or it may continue through the entire systole; its point of maximum intensity is usually directly over the apex (transmitted through the contracted left ventricular musculature); it is often transmitted to the axilla and the angle of the scapula (which is not of much importance diagnostically); rarely it is heard best along or near the left edge of the sternum, or in the fourth or the third, sometimes the second, interspace (pulmonary area)—in consequence of the direction of the blood current and of the auricle being in close contact with the chest; rarely it is heard best in the back between the scapulæ; it is often loudest when the patient is recumbent; sometimes it is heard only when he is recumbent. The pulmonic second sound is accentuated, sometimes reduplicated. The pulse during compensation is not characteristic; it is full and regular, although perhaps of low tension. Occasionally in extreme insufficiency with considerable ventricular hypertrophy a pulmonary pulsation, a diffuse systolic expansion of the lungs (analogous to the liver pulsation of tricuspid insufficiency), may be palpable. With failure of compensation, the pulse becomes small, frequent, and irregular; the physical signs of cardiac dilatation assume the ascendency; the systolic murmur changes in character, lessens in intensity or disappears entirely, or perhaps is audible only during the stronger systoles that characterize the arrhythmia; the pulmonary second sound loses its accentuation, and the signs of relative tricuspid insufficiency, general venous engorgement, and œdema develop.

Diagnosis.—The characteristic diagnostic signs consist of enlargement of the right ventricle, a systolic thrill and a systolic murmur at the apex, and an accentuated pulmonic second sound. Relative mitral insufficiency is to be differentiated from organic insufficiency, not by the physical signs, which may be alike in both, but by the antecedent conditions and associated phenomena, by the presence of an adequate causative factor to give rise to dilatation of the heart and a relative mitral insufficiency, and by the disappearance of the murmur with improvement in the condition of the heart (lessening of the area of dulness, and of dilatation, and increase of vigor). The systolic murmur of aortic stenosis is sometimes very loud at the apex, but it is loud also at the aortic area and is propagated into the carotid artery, and it is associated with the other distinctive signs of aortic stenosis. Difficulty is not infrequently experienced in differentiating so-called functional, inorganic, accidental, or hemic murmurs, as well as certain exocardial, cardiorespiratory, sounds. While it is true that the so-called functional murmurs are usually soft and blowing in character, are heard best, as a rule, in the

pulmonary area, and are not far transmitted, they often of themselves are not distinguishable from organic murmurs; the diagnosis is to be based less upon the character of the murmur than upon the concomitant physical signs, such as hypertrophy of the heart and accentuated sounds, on the one hand, and fever, anemia, etc., on the other hand; and the disappearance of the murmur with the subsidence of the fever, anemia, etc. The cardiorespiratory murmurs are usually heard along the left border of the heart, are soft and blowing in character, often loudest at the height of inspiration, and disappear when the patient stops breathing. In most cases of valvular lesion, in fact, one may elicit almost if not quite conclusive evidence of the valvular disorder without any attention to the presence or absence of a murmur whatever.

Mitral Stenosis.—Mitral stenosis occurs in about 16 per cent. of the cases of valvular disease, and double mitral disease in about 12 per cent., so that mitral obstruction occurs in a total of 28 per cent.

Etiology.—Mitral stenosis may be due to: (1) Endocarditis, in which event it is especially common in adolescents, and young adults, as a sequel of rheumatism, "growing pains," chorea, scarlatina, and other infections; and (2) slowly progressing sclerosis, analogous to arteriosclerosis, a form encountered in later life, and in men as well as in women. In many cases, especially in adult women, no etiological factor can be determined with certainty—which has led some observers, perhaps too frequently, to believe them congenital. Two forms of the disease may be distinguished: in the one, found especially in young persons, the leaflets, perhaps only slightly thickened, have become adherent at their edges, and the chordæ tendineæ somewhat thickened, shortened and fused, so that the valve viewed from the auricle seems to have become converted into a hollow cone—the so-called funnel-shaped mitral valve. In the second variety, more common in adults, the leaflets are much thickened, indurated, perhaps calcified, and distorted, and are represented by a rigid, often atheromatous, mass of tissue exhibiting a central linear slit, admitting perhaps only the tip of the little finger—the so-called button-hole mitral valve. The mitral ring is often converted into a fibrous or calcareous ring, the chordæ muscles are shortened and thickened, the papillary muscles even may be inserted directly into the edge of the leaflets. In both varieties there is practically always an associated mitral insufficiency; rarely perhaps in the first-mentioned variety, when the leaflets are pliable, there may be a pure mitral stenosis. (3) A relative obstruction (or stenosis) is occasionally produced by exuberant vegetations, ball-valve thrombus, aneurysmal dilatation of the leaflets, etc., and a condition sometimes spoken of as relative stenosis ensues when the cavities become much dilated, while the orificial ring remains normal or is relatively slightly enlarged.

Pathological Physiology.—In mitral stenosis the morbid phenomena are much like those in mitral insufficiency. There is a similar increase in the intra-auricular pressure toward the end of auricular diastole, due to the blood from the lungs flowing into an insufficiently emptied

auricle; the consequent dilatation and stretching excite the auricle to very vigorous contractions, which become augmented in consequence of the obstruction to the discharge of blood. The result is an extraordinary enlargement (hypertrophy and dilatation) of the left auricle, which may attain dimensions three or four times the normal (to 200 c.c. or more). Increased intrapulmonary pressure is followed by hypertrophy and dilatation of the right ventricle, which as in mitral insufficiency is the efficient factor in maintaining compensation. In some cases, for a time at least, the increased power of the left auricle serves to supply a normal amount of blood to the left ventricle, which in consequence presents no noteworthy deviations from the normal. In many cases, however, on account of associated mitral insufficiency, the left ventricle is somewhat enlarged; in most cases the amount of blood supplied to the left ventricle is less than normal, in consequence of which it is often said to become reduced in size (this is more apparent than real). Perhaps in primary or uncomplicated mitral stenosis the left ventricle does lessen in size, but the tendency in this direction is usually counteracted by the primary and usually associated mitral insufficiency. The final breakdown and the visceral and other consecutive changes are similar to those that develop in mitral insufficiency.

Symptoms.—The disease is not incompatible with many years of life and comparative freedom from distress. Symptoms when they do develop are not to be sharply differentiated from those of mitral insufficiency, although they are likely to be more marked or more readily provoked. Cyanosis usually is of a higher grade, and often gives rise to a peculiar "mitral facies" that of itself suggests the diagnosis; dyspnœa is more readily induced by exertion and excitement; hemoptysis is more frequent and more easily provoked; and attacks of bronchitis are more prone to develop. Precordial pain and distress is often an obstrusive and sometimes an intractable symptom. Recurring endocarditis is quite common, and may be the cause of otherwise inexplicable attacks of fever and leukocytosis. The vegetations disintegrating are often carried into the circulation and set up embolism in different parts of the body, notably in the brain, where they may give rise to aphasia, hemiphlegia, etc.; but also elsewhere. Paralysis of the left vocal cord (simulating aneurysm) occasionally results from pressure on the recurrent laryngeal nerve by the dilated left auricle. The disorder developing in early life may lead to stunting of the bodily growth. Recurring attacks of failure of compensation are perhaps more common than in mitral insufficiency. Compensation usually becomes reëstablished, but eventually more or less complete failure ensues, and the decompensation phenomena mentioned in connection with mitral insufficiency develop.

Physical Signs.—The signs of hypertrophy of the right ventricle are the same as those in mitral insufficiency (which is often associated and presents its own diagnostic signs). In mitral stenosis, perhaps, there is more enlargement of the right ventricle; the left auricle also is usually more enlarged, and may perhaps present some percussion evidence of

this in dulness upward and to the left; but commonly this is not found, since the increase in the size of the left auricle is usually predominantly anteroposterior. Occasionally there is impaired resonance in the left infraclavicular fossa, due perhaps to compression of the lung by the dilated left auricle; in other cases the enlarged auricle may occasion some dulness in the left interscapular region. At the apex a diastolic or presystolic thrill is commonly palpable, and usually followed by, or the thrill ends in, a quick, thumping, or tapping apex beat. The murmur of mitral stenosis, which is often limited to a small area near the apex, but is not infrequently audible over a considerable area about the apex, is diastolic in time; it may occur during any part of the diastole or throughout the entire diastole. Perhaps the commonest is the interrupted diastolic murmur (so-called Fraentzel murmur), a murmur louder at the beginning and the end of diastole than during the mid-period (a mid-diastolic diminuendo and presystolic crescendo murmur); this is due doubtless to the greater quickness of the flow of blood at the beginning of diastole than during the mid-period, and an increased acceleration at the end of diastole occasioned by the contraction of the auricle. The next most common murmur is the definitely late-diastolic (or presystolic) rough, rumbling, crescendo murmur; then a simple diastolic murmur, or a continuous murmur occupying the entire diastole and the long pause. There may also be a double diastolic tone at the apex (*bruit de rappel,* Bouillard), which under excitement may be replaced by a double murmur, or a sound and a murmur. A loud, sharp, snappy first sound at the apex is quite characteristic of mitral stenosis, and in the absence of a murmur (but associated with the other signs) suffices for the diagnosis. It is best explained as due to differences greater than the normal in the tension of the mitral leaflets at the beginning and the end of systole (closure-time). These auscultatory signs vary much from time to time, being much influenced by quietness and excitement and by strength and weakness of the heart muscle. The pulmonary second sound is accentuated or reduplicated. The pulse is small and irregular, due to incomplete filling of the ventricle. When the heart fails the murmur may disappear (auricular paralysis) and the second sound at the apex become inaudible, so that only the loud first sound may be heard at the apex; to which, however, are usually added the signs of tricuspid regurgitation, and the general evidences of failure of the circulation. With restoration of compensation the murmur usually again becomes audible, and the pulmonic second sound accentuated.

Diagnosis.—The characteristic diagnostic signs consist of enlargement of the right ventricle (and also of the left auricle, if it can be demonstrated), a diastolic or presystolic thrill and murmur at the apex, a loud, snappy, first sound at the apex, an accentuated pulmonic second sound, and a small, weak, and irregular pulse. Aortic insufficiency accompanied by a presystolic (Flint) murmur is to be distinguished, not by the character of the murmur, but by the associated

physical signs. Adherent pericardium, with dilatation of the heart, also is sometimes accompanied by a presystolic murmur—which, however, rarely if ever has a crescendo character, and is not accompanied by the other physical signs of mitral stenosis, but by those of adherent pericardium. The presystolic murmur of the very rare tricuspid stenosis will scarcely lead to diagnostic confusion.

Aortic Insufficiency.—Aortic insufficiency occurs in about 36 per cent. of the cases; in about 12 per cent. the lesions are described as double aortic disease, and in 18 per cent. there is associated mitral valve disease.

Etiology.—Aortic insufficiency may be due to: (1) Endocarditis. This occurs especially in adolescents and young adults, and although uncommon, is perhaps more common than is generally believed. It may be a sequel of rheumatic fever, but is more likely to result from pyococcic, pneumococcic, and gonococcic infections. In perhaps a few cases the vegetations disappear without permanently damaging the leaflets. Usually, however, one or more of the leaflets becomes thickened, indurated, and curled upon itself, so that the normal approximation during diastole is impossible. In a few cases the leaflets become adherent to one another at their adjacent edges, so that to insufficiency, stenosis is added. Rarely a leaflet may become perforated by the necrosing or ulcerative process. (2) Arteriosclerosis. This is the common form. Although it occasionally occurs in women, it is especially frequent in men after the fortieth year, and is associated with antecedent alcoholism, syphilis, and excessive muscular strain, as well as the causative factors of arteriosclerosis and high arterial tension. Occasionally following syphilis, the disorder may develop in comparatively young men—several years after the infection. In later life a so-called parasyphilitic form occurs and is rather frequently associated with tabes dorsalis. The lesions are those of arteriosclerosis (involving especially the aorta), of which the valvular disorder constitutes a part. One or more of the valve segments lose their tonicity and plasticity, become indurated, curled at their edges and consequently shortened, and are then unable to close the orifice. In some cases calcification follows and the segments become rigid; or they become adherent at their adjacent edges, so that to the insufficiency stenosis is added; but stenosis is much less common than is usually thought. (3) Rarely the disorder may be traumatic, developing in consequence of sudden undue muscular strain, severe contusions or compression of the chest, etc., which lead directly to rupture of usually a single leaflet (normal or previously diseased). (4) Relative insufficiency rarely ensues in consequence of dilatation of the aortic ring, due to loss of elasticity, and associated with considerable dilatation of the arch of the aorta. (5) In a few cases the condition is congenital, due to fusion or other malformation of the leaflets.

Pathological Physiology.—In consequence of incompetency of the aortic valve the left ventricle during diastole receives blood from two sources—the normal volume from the left auricle and a variable amount,

the regurgitating stream, from the aorta. Whether the regurgitating stream is considerable in amount, and gradually increases with the progress of the disorder, as is generally believed, or is only a relatively small amount of the total volume of ventricular blood, as the recent researches of Stewart suggest, the fact is that the intraventricular diastolic pressure becomes increased. The tension and stretching to which the ventricular musculature is thus subjected stimulate it to vigorous contractions, in an effort to expel the augmented amount of blood, and the constant recurrence of the stimulus ultimately induces more or less hypertrophy. The enlargement of the cavity of the ventricle (dilatation) and the concomitant hypertrophy may and usually do counterbalance each other, so that for a considerable period the periphery is sufficiently supplied with blood and the patient suffers no noteworthy ill consequences. Ultimately the heart may reach enormous proportions, the largest seen (bovine heart), and may weigh 1200 grams or more. In the course of time dilatation assumes the ascendency over the hypertrophy in consequence of deficient blood supply through the coronary circulation (narrowing of the coronary orifices and sclerosis of the vessels in their course) and concomitant myocardial degeneration; the equilibrium of the circulation is no longer maintained and failure of compensation ensues. The gradual enlargement of the ventricular cavity leads to relative mitral insufficiency, and this in turn to its own ill effects, already mentioned; organic mitral insufficiency also may occur. The aorta, especially the ascending arch, is usually considerably dilated (so-called fusiform aneurysm), but it may be only unusually distensible; that is, there are signs of enlargement during life (expansion due to the systolic output of a large volume of blood), but no material increase in size after death (so-called dynamic dilatation). The systolic output of a large volume of blood is also apparent in the violent throbbing of the superficial arteries, which, as the result of the long continued strain to which they are subjected, usually reveal more or less sclerosis (which may be due also to other factors—alcoholism, syphilis, plumbism, etc.). In the acute cases that develop suddenly in consequence of rupture or rapid destruction of a leaflet, compensation cannot be immediately effected, and the patient suffers immediate distress. An old sclerotic valve may become the seat of acute endocarditis.

Symptoms.—Well compensated aortic insufficiency may be disclosed only by a physical examination of the heart; indeed, in many cases the disorder is for a long time overlooked on account of the absence of subjective symptoms, the slight degree of hypertrophy of the left ventricle, and the softness or indistinctness of the murmur. In many cases the earliest symptoms consist of dizziness, perhaps amounting almost to faintness, throbbing headache, flashes of light before the eyes, tinnitus aurium, muscular weakness, and palpitation of the heart and dyspnœa on sudden exertion. The phenomena, which, as a rule, rapidly subside upon the restoration of quiet, are attributable to cerebral anemia. Marked pallor (aortic facies) and a readily induced flush are usually

conspicuous symptoms; the flush may be accompanied or followed by sweating, which with occasional hemoptyses has suggested tuberculosis to the unwary; there may also be slight œdema due to the anemia. Precordial pain is often present and sometimes distressing; it is usually dull and aching, but may be severe and sharp and radiate to the neck or arms (especially the left). True angina pectoris also occurs; more frequently in aortic insufficiency than in any other form of valvular disease.

As compensation fails the aforementioned symptoms become persistent and aggravated: cough, dyspnœa, and palpitation of the heart in a rather pallid subject are quite characteristic. With the advent of relative mitral insufficiency the symptoms of this condition may supervene; but cyanosis is rarely marked, hemoptysis is not common, and œdema is not extreme or widespread, being usually limited to the feet and ankles and other dependent parts of the body. Embolic phenomena are not uncommon—in the brain, spleen, kidneys, etc. Nervous symptoms are often obtrusive—irritability, restlessness, insomnia, melancholia, delusions, suicidal tendencies, etc. For a long time compensation may be unstable, and although when more or less lost it may sometimes be restored, the restoration is much less readily effected than in any other form of valvular disease; this decompensation, especially in elderly subjects, often betokens the final lethal termination. Sudden death is not uncommon, being often due to blockage of the coronary arteries.

In the acute cases that develop from rupture (traumatism or ulcerative processes), dyspnœa, precordial pain, palpitation of the heart, sometimes hemoptysis, etc., often come on immediately and may prove extremely distressing, until the heart finally more or less effectually compensates the lesion.

Physical Signs.—Depending upon the youth of the subject when the disorder developed and the degree of hypertrophy of the left ventricle, there is more or less bulging of the precordium; the cardiac impulse is forcible and widespread, and the apex beat (the palpable beat farthest downward and outward) is displaced to the sixth, seventh, or eighth intercostal space outside the mamillary line to the anterior or midaxillary line; it is usually strong and forcible and heaving in character, and, considering the size of the heart, is rather pointed (in contrast to the much wider and rounded apex of hypertrophy of the right ventricle). Visible and forcible pulsation of the arteries is present during compensation and is quite significant of aortic insufficiency; it is especially obvious in the aorta (episternal notch and perhaps the second right intercostal space), the carotids, temporals, retinals, subclavians, brachials, radials, femorals, and often the small arteries of the hands and feet. The pulsations may be so forcible as to occasion a visible nod of the head with each heart beat (Musset's sign) and a jerk to a leg crossed over the other leg. In cases of marked elongation and tortuosity of the arteries, the pulsations are peculiarly serpentine, and were interpreted

by an alcoholic patient of mine as certain evidence of snake invasion. Usually the pulsation is transmitted to the capillary vessels, and may be apparent upon mere inspection of the face or hands, or the oral mucous membrane (uvula, for instance); or it may be readily brought in evidence by rubbing the skin for a moment, or by slight pressure on a finger nail, or on the lip mucous membrane by a glass slide. This is significant but not diagnostic of aortic insufficiency, since it may be found also in other conditions of relaxation of the peripheral arteries (in health as well as in disease). In a few cases there may be an arterial pulsation in the liver. In a few cases the pulsations are still further transmitted—into the small veins, especially of the back of the hands (the penetrating or centripetal pulse of Quincke). The arterial pulse on palpation is quite characteristic—of increased volume and force, sudden of impact and of fall, and is empty between beats—the so-called Corrigan, water-hammer, pistol, or receding pulse; pulsus altus et celer. Usually it may be well perceived by grasping the muscle of the forearm in the hand. The sudden collapse is usually attributed to diastolic regurgitation of a large volume of the blood through the incompetent aortic valve, but it is more likely due in large measure to reflex dilatation of the peripheral arterioles, induced by stimulation of the ventricular wall in consequence of increased pressure (Stewart); and to this also is due the capillary pulse and the penetrating venous pulse. Stewart interprets this peripheral vascular relaxation as a means of preventing the ill effects of undue pressure on the ventricle. A diastolic thrill is sometimes palpable at the base, near or beneath the sternum; rarely it may be felt also at the apex; occasionally a thrill at the apex is presystolic (analogous to the Flint murmur); in many cases there is a double thrill at the base, the systolic element being well transmitted into the aorta and the carotids; this is by no means always due to associated aortic stenosis.

On percussion there is increase of the cardiac dulness diagonally, in the direction of the long axis of the heart. The area of dulness becomes diagonally ovoid, downward and to the left; only in advanced cases with consecutive dilatation and relative mitral insufficiency does the dulness become much increased to the right. The characteristic murmur is diastolic in time, soft and blowing in character, beginning with diastole and gradually decreasing in intensity; it is heard best usually (but not always) over the middle or at the left edge of the sternum on a level with the third rib, and it is transmitted down the left edge of the sternum to the ensiform cartilage, or to the apex (where it may be quite as loud as at the base of the heart), or to the axilla (apart from its transmission to the apex). Sometimes the murmur is so faint as to be almost if not quite inaudible; it may be harsh and rasping, or musical. In most cases there is a double murmur in the aortic area (the to-and-fro bellows murmur of Stokes)—the diastolic murmur already mentioned, and a systolic murmur often harsh in character, and transmitted into the carotids; it is due to roughening of the aortic leaflets, sclerosis of the aorta, dilatation (fusiform aneurysm) of the aorta, aortic stenosis (rare),

true aneurysm, or the impact of the regurgitating with the outgoing stream of blood. In many cases (25 to 50 per cent.) a presystolic (Flint) murmur, quite like that of mitral stenosis, is audible at the apex; it is probably due to the regurgitating stream of blood impinging upon and actually driving the anterior mitral leaflet toward the centre of the ventricle, thus interfering with the entrance of blood into the ventricle at the time of auricular systole by producing essentially a moderate mitral stenosis (Guiteras); or to vibrations of the anterior mitral leaflet between the two streams of blood. The aortic second sound is usually wholly, occasionally only partly, replaced by the diastolic murmur (perhaps the lesion is less serious when the sound is only partly replaced). The systolic murmur (sometimes both murmurs) is more or less well transmitted to the subclavian, axillary, and carotid arteries. A rather loud, sharp, systolic (vascular diastolic) sound (pistol-shot sound) is sometimes audible over the larger arteries, especially the femorals; it is produced locally and is not transmitted from the heart. Rarely two sounds may be heard, the second coincident with the collapse of the artery (Traube's sign). Pressure with a stethoscope on the femoral artery, in addition to a systolic murmur thus normally produced, sometimes results in the production of a cardiac diastolic murmur (Durosiez's sign). Systolic contraction of the pupil (due to elongation of the vessels of the iris) and diastolic dilatation (lessened amount of blood) may be observed (Landolfi's sign).

With the development of myocardial weakness, dilatation of the heart, relative mitral insufficiency, etc., the physical significance of these disorders also develop.

Diagnosis.—The characteristic diagnostic signs consist of evidences of enlargement of the left ventricle, a diastolic murmur at the base of the heart transmitted down the sternum, toward the apex beat, or into the axilla, marked visible pulsations of the superficial arteries, the Corrigan pulse, a capillary pulse, and Traube's and Durosiez's signs. The murmur is often very faint or quite inaudible, in which event the diagnosis may be quite apparent from the hypertrophy of the left ventricle, the collapsing pulse, and the pallor of the patient. The commonly associated systolic murmur in the aortic area should be correctly interpreted; usually it does not mean aortic stenosis. The presystolic (Flint) murmur at the apex is to be distinguished from the similar murmur of mitral stenosis by the associated signs, especially the presence of hypertrophy of the left rather than the right ventricle, absence of accentuation of the pulmonic second sound, the presence of arteriosclerosis, and the character of the pulse. It is scarcely likely that the rare inorganic diastolic murmurs, or those due to pulmonary insufficiency, will lead to diagnostic confusion. Evidences of fusiform dilatation of the aorta must not be mistaken for true aneurysm.

Aortic Stenosis.—Aortic stenosis alone occurs perhaps in 1 to 2 per cent. of the cases of valvular disease; in about 12 per cent. of the cases the condition is described as double aortic disease (insufficiency and stenosis).

Etiology.—Aortic stenosis may be due to: (1) Endocarditis. This is quite rare, but may follow rheumatism and other infections, and occurs in young subjects. In consequence of organization of the vegetations, the leaflets become thickened, indurated, and adherent at their adjacent margins, so that varying grades of obstruction ensue. Later a deposit of lime salts may occur, so that the leaflets with the aortic ring may form a dense calcified mass of tissue with a variable sized and shaped opening. (2) Arteriosclerosis. This is the common form of aortic stenosis; but in itself it is rare, and is always part of general arteriosclerosis, especially of the aorta. It occurs, therefore, especially in elderly subjects. The process is slow, although usually progressive, and ultimately leads to thickness and marked, often cartilaginous, rigidity of the leaflets, with corresponding obstruction to the blood curent. In advanced cases there is usually considerable calcareous infiltration, in which event the lesions are much like those mentioned in connection with the endocarditic form. (3) Relative aortic stenosis (a misnomer) is a term sometimes used to designate that condition in which, with normal aortic leaflets and aortic ring, the aorta just beyond is unusually dilated. (4) There are other rare types: fusion and thinning of the leaflets—perhaps congenital; fibrocalcareous thickening and induration of the aortic ring without involvement of the leaflets; a subvalvular constriction involving the mitrosigmoidian sinus and probably due to fœtal endocarditis, etc.

Pathological Physiology.—On account of obstruction at the aortic orifice, the left ventricle is unable to expel the normal amount of blood in the normal time. In consequence, a residual amount of blood is present in the ventricle at the beginning of diastole, which, with the normal amount received from the auricle, leads to increased intraventricular pressure; this, together with stimuli occasioned by resistance at the aortic orifice to be overcome, induces hypertrophy of the left ventricle —which is the effective factor in maintaining compensation, although it never attains the dimensions of that of aortic insufficiency. This hypertrophy is often sufficient to enable the heart to propel the normal amount of blood into the aorta; no noteworthy dilatation ensues, and the patient suffers no ill consequences. The systole, however, is prolonged. When the stenosis becomes greater than can be compensated by hypertrophy of the left ventricle, or when the myocardium becomes enfeebled in consequence of insufficient nourishment (coronary sclerosis), dilatation ensues, and ultimately perhaps relative mitral insufficiency and its consequences.

Symptoms.—Well compensated aortic stenosis is often for a long time latent. Symptoms when they develop are much like those of aortic insufficiency, which is usually associated; with absence of aortic insufficiency the symptoms are likely to be less severe, although perhaps readily induced by exertion. When dilatation ensues, the symptoms of failing compensation and relative mitral insufficiency develop.

Physical Signs.—In young subjects there may be bulging of the precordium due to hypertrophy; but since the hypertrophy is often slight,

for a long time it may be overlooked, especially in elderly emphysematous subjects. The signs of hypertrophy when apparent are analogous to those mentioned in connection with aortic insufficiency, although less marked. Usually there is a rough systolic thrill at the aortic cartilage propagated into the aorta and the carotid, perhaps other nearby arteries. The pulse is quite characteristic: it is small or moderate in volume, slow (pulsus tardus), and rises slowly (prolonged systole), is well sustained (broad summit), and falls slowly. On auscultation there is a systolic murmur at the base of the heart, which obscures or replaces the first sound and is usually rough and rasping in character, sometimes loud and musical (perhaps audible at a distance), but may be soft (especially when the ventricle is weak); its point of maximum intensity is at or near the aortic cartilage, and it is usually transmitted to the carotid arteries, and sometimes to the descending aorta (audible in the left interscapular space), the axillary arteries, etc. Occasionally it is very loud at the apex (without any associated mitral insufficiency). The second aortic sound is commonly absent or weak, or replaced by a diastolic murmur, on account of rigidity of the aortic leaflets and commonly co-existing aortic insufficiency. With the advent of myocardial weakness and dilatation, relative mitral insufficiency usually ensues and materially modifies the previously existing physical signs.

Diagnosis.—The characteristic diagnostic signs consist of evidences of enlargement of the left ventricle, a systolic thrill and a systolic murmur at the base of the heart and transmitted into the aorta and carotid arteries, a weak or absent aortic second sound, and a slow (as distinguished from an infrequent) pulse. A basal systolic murmur, upon which the diagnosis is often based, is usually due to some other disorder, such as roughening of the aortic leaflets, sclerosis of the aorta, dilatation (fusiform aneurysm) of the aorta, true aneurysm, or the impact of a regurgitating with the outgoing stream of blood. Sclerosis or atheroma of the aorta may be differentiated by absence of the characteristic pulse and usually also of a thrill, and the presence of an accentuated aortic second sound and of other signs of arteriosclerosis. Dilatation of the aorta and true aneurysm may be recognized by their own diagnostic signs. The basal systolic murmurs of anemia, fever states, etc., usually have an obvious cause, are rarely loudest in the aortic area, and do not give rise to enlargement of the left ventricle. Pulmonary stenosis and mitral insufficiency, which perhaps might enter into the diagnosis, give rise to enlargement of the right rather than the left ventricle, and other unequivocal signs.

Tricuspid Insufficiency.—Etiology.—Tricuspid insufficiency rarely results from acute or chronic endocarditis (antenatal or postnatal). Usually the condition is relative and due to inability of the muscle of the right heart to perform the work to which it is subjected. This insufficiency, especially as a temporary disorder, is much more common than is suspected; it develops in health under the stress of unusual or sudden strain (athletic exercises, parturition, etc.), and in disease when

the enfeebled myocardium is suddenly called upon for some extra work—in both of which circumstances the safety-valve action of the right ventricle (involving the wall of the ventricle, the papillary muscles, which readily become fatigued and act imperfectly or ineffectually, and the tricuspid ring) becomes operative and prevents serious and lasting damage to the myocardium. The more obvious and clinically important tricuspid insufficiency occurs in chronic hypertrophy with consecutive dilatation of the right ventricle, which in turn results from advanced mitral valve disease, chronic diseases of the lungs (emphysema, bronchitis, interstitial pneumonitis, pleural adhesions, etc.), pericardial adhesions, and myocardial weakening due to any local or general cause. There is associated congestion of the liver, spleen, kidneys, gastro-intestinal tract, etc., œdema of the subcutaneous tissues, effusions in the serous sacs, etc.

Symptoms.—The symptoms are those of failing cardiac compensation and are due to the associated passive congestion and consequent functional inactivity of the organs. The embarrassment of the heart itself is obvious in precordial distress or pain and palpitation of the heart. Congestion of the lungs leads to cyanosis, dyspnœa (aggravated upon exertion and excitement), cough, expectoration, hemoptysis, etc.; of the liver, to local pain and tenderness; of the gastro-intestinal tract, to poor appetite, disordered digestion, flatulence, nausea, vomiting, constipation, etc.; of the kidneys, to oliguria, albuminuria, cylindruria, erythrocyturia, etc. Œdema is always present and may become universal; effusions are common in the serous sacs, especially the right pleura; ascites is rarely present before general œdema, but it may be disproportionately marked.

Physical Signs.—The physical signs consist of dilatation of the heart, especially of the right side (the dulness may extend to the right nipple line); a systolic murmur (usually soft and blowing, but sometimes rough) in the tricuspid area (and especially significant if heard to the right of the sternum, where the murmur of mitral insufficiency is rarely heard); a ventricular systolic autochthonous pulsation in the veins of the neck, often especially marked in the jugular bulb and sometimes propagated to adjacent veins (rarely a murmur may be heard); and a ventricular systolic expansile pulsation of the liver. The signs of the antecedent disorder, usually mitral valve disease, or chronic disease of the lungs, etc., are also present. Enfeeblement of a previously accentuated pulmonic second sound is a valuable sign of the development of tricuspid insufficiency; as the insufficiency grows less (or the right side of the heart grows stronger) the pulmonary second sound again becomes accentuated. The safety valve leakage usually occasions no noteworthy symptoms and soon subsides; indeed, the occurrence of the leakage probably prevents the onset of more or less serious symptoms.

Tricuspid Stenosis.—**Etiology.**—Tricuspid stenosis is a rather rare condition. It may be due to endocarditis, either antenatal or postnatal (usually rheumatic); rarely it seems to be primarily degenerative, analo-

gous to the arteriosclerotic process. It is most common in women (75 per cent. of the cases), and in more than 99 per cent. of the cases is associated with disease of one or more of the other valves, especially mitral stenosis (56 per cent. of the cases). The characteristic lesions consist of hypertrophy and dilatation of the right auricle.

Symptoms.—The symptoms are virtually those of mitral stenosis. Cyanosis is often the earliest obtrusive symptom, and may be present for months before there are other noteworthy manifestations of decompensation. Polycythemia is common and the fingers may be clubbed. When the heart begins to fail, the symptoms cannot be distinguished from those of tricuspid insufficiency. Dropsy may occur early and is unusually persistent.

Physical Signs.—The physical signs are commingled with those of the complicating lesions; but the significant signs consist of increase of the cardiac dulness to the right (which may extend to or beyond the mammillary line); a presystolic or diastolic thrill, and a usually rough, rumbling presystolic or diastolic murmur, sometimes ending in a rather sharp first sound, at the end of the sternum; and presystolic (auricular systolic) pulsations in the veins of the neck and in the liver.

Pulmonary Insufficiency.—Etiology.—Pulmonary insufficiency is very rare as an acquired affection, but it sometimes results from acute endocarditis (usually set up by the pyococci, sometimes by the gonococcus). Occasionally in cases of long-standing high pulmonary tension there is some sclerosis of the leaflets and consequent insufficiency. Perhaps relative insufficiency sometimes results from stretching of the pulmonary ring in conditions of high pulmonary pressure. The lesions much resemble the analogous lesions of aortic insufficiency, involving, however, the right instead of the left ventricle.

Symptoms.—Symptoms are in abeyance until the right heart begins to fail, whereupon the phenomena common in decompensation develop.

Physical Signs.—The physical signs consist of hypertrophy and later of dilatation of the right ventricle, and a diastolic murmur at the base of the heart, usually loudest in the pulmonary region and transmitted down the left edge of the sternum. Aortic insufficiency may be excluded by the absence of enlargement of the left ventricle and of the characteristic vascular phenomena.

Pulmonary Stenosis.—Etiology.—Acquired pulmonary stenosis is the rarest of valvular lesions. Occasionally it follows acute endocarditis, or the endocarditis may become engrafted upon an old, perhaps congenital defect; occasionally also the lesion may be due to slow sclerosis, when it is associated with pulmonary endarteritis. Very rarely, perhaps trauma may lead to rupture of one of the leaflets. The lesions are much like the analogous aortic stenosis, involving, however, the right instead of the left ventricle.

Symptoms.—Symptoms are not obtrusive until the right heart begins to fail, whereupon the symptoms common to decompensation ensue.

Physical Signs.—The physical signs consist of hypertrophy and dilatation of the right ventricle, a systolic thrill and murmur in the pulmonary area, and a weak or absent pulmonary second sound; or the sound may be replaced by a diastolic murmur. A systolic murmur is very common in the pulmonary region, but it is rarely due to pulmonary stenosis: it may occur in healthy, thin-chested persons, especially children, and is often induced by exercise; it is common in anemic, cachectic, and fever states; it may be due to the interaction of the heart and the lung (cardiopulmonary murmur); and it may be due to aortic stenosis (and the other factors mentioned as causative of an aortic systolic murmur), or to mitral insufficiency. In most of these cases the diagnostic inference is obvious: aortic stenosis may be excluded by absence of hypertrophy of the left ventricle, of propagation of the thrill and murmur into the carotid arteries, and of the characteristic pulse. Mitral insufficiency may be excluded by the absence of the common signs, especially the accentuated pulmonary second sound.

Combined Valvular Lesions.—In almost, if not quite, one half of the cases of valvular disease, there is more than a single lesion; that is, there may be insufficiency and stenosis at one valve, or two or more valves may be diseased. This double or multiple involvement of the valves may develop at the one time, or, what is more common, involvement of one valve follows that of another; and the lesions may be alike in nature or dissimilar. Thus, while rheumatic and other infections commonly involve only the mitral valve, they not infrequently at or about the same time, especially in children, involve also the aortic valve, and occasionally also one or both valves of the right side of the heart. More commonly, however, the primary lesion, usually insufficiency, is followed in the course of time by cicatrization and consequent stenosis: thus mitral insufficiency often in the course of time becomes associated with stenosis (so-called double mitral disease); mitral stenosis is rare as a single lesion, being usually associated with insufficiency; aortic insufficiency is often the primary and may remain the only lesion, but sometimes becomes associated with stenosis (double aortic disease); aortic stenosis as a single lesion is the rarest of all in the left side of the heart. The developing stenosis may for a time at least prove beneficial in lessening the primary insufficiency and retarding the development of its ill effects. Varying combinations of aortic and mitral valve disease also occur, especially aortic and mitral insufficiency, due usually to sclerosis rather than to endocarditis. The commonest combination of valvular disorders, however, is that of an organic lesion at one orifice, followed by relative insufficiency at another: mitral insufficiency or stenosis followed by tricuspid insufficiency, and aortic insufficiency or stenosis followed by relative mitral insufficiency and later relative tricuspid insufficiency.

Physical Signs.—Associated valvular lesions materially alter the physical signs of individual lesions and proportionately increase the diagnostic difficulties. In the diagnosis accuracy will be best achieved by

paying less attention to the murmurs than to the other physical signs, and by postulating the fewest defects compatible with an explanation of the recognizable physical signs. A systolic murmur alone is of no value whatever in the diagnosis; a diastolic murmur almost certainly means a valvular defect, but in this event there are always corroborative signs. The loudest, most obtrusive murmur (usually systolic) may be due to a secondary lesion (relative insufficiency), the murmur of the primary lesion being soft or almost inaudible, but in this event also the concomitant physical signs correctly determined point the way to the correct interpretation of the phenomena.

Diagnosis.—The diagnostic criteria of the different forms of valvular disease have been sufficiently dwelt upon. But the diagnosis is by no means complete with the naming of the valvular defect; it must be much more comprehensive, and should include the nature of the valvular disorder (whether endocarditic, sclerotic, or relative; obsolete or active), the degree and adequacy (stability) of the compensating mechanism, and the factors that tend to or actually provoke decompensation in the individual case; in other words, in addition to ascertaining the valvular defect one must determine also the functional capacity of the myocardium —the relative preponderance of hypertrophy or dilatation, and the ability of the heart to do a measured amount of work without subjective or objective distress.

Prognosis.—The prognosis in valvular disease of the heart depends in part upon the nature and severity of the valvular defect, but in large measure upon the ability of the myocardium to effect and maintain compensation, which in turn is much dependent upon the patient's general health and mode of life. The anatomical valvular defects, of course, cannot be cured, although material benefit often follows the supervention of stenosis upon a well-marked insufficiency; relative defects (insufficiencies) due to stretching of orifices may entirely disappear if the affected orifices regain their normal tone. The anatomical defects, however, are not incompatible with long life and more or less freedom from distress. In point of gravity, the mitral lesions are less serious than the aortic, the order from the least to the most serious being, mitral insufficiency, mitral stenosis, aortic stenosis, and aortic insufficiency. The cases associated with extensive arteriosclerosis (usually aortic insufficiency) are more grave than those the result of past rheumatism, since there is usually also sclerosis of the coronary arteries with its attendant danger of angina pectoris, thrombosis and embolism of the coronary vessels, and consequent sudden death, etc. In the aortic cases also compensation, when once disturbed, is much less readily restored than in the mitral cases, and when restored it is usually unstable. Double or triple valvular defects, whether organic or relative, add materially to the gravity of the prognosis. In children the prognosis is generally bad; in women it is better than in men, because of their quieter life, the preponderance of mitral lesions, and their less addiction to the vices of intemperance, venery, and hard manual labor. Pregnancy,

however, adds materially to the gravity of the condition, although a considerable number of cardiopaths bear and rear large families. In mitral cases there is rarely associated pulmonary tuberculosis (due to relative immunity afforded by chronic passive congestion).

Treatment.—It seems quite likely, although difficult to prove, that chronic valvular disease of the heart may be prevented in some cases, or that its serious consequences may be lessened or retarded. Since most of the cases of the endocarditic group are due to rheumatism, the prompt and efficient jugulation of this infection should tend to minimize its complications: that this really occurs there can be no doubt, although from the nature of the disorder definite proof can scarcely be adduced. Since the cases of the sclerotic group are associated with arteriosclerosis, and since arteriosclerosis is largely the penalty of unwise living and divers sorts of indiscretions, it is quite probable that a rational and hygienic mode of life, tending to retard the degenerative changes of the arteries, should tend also to prevent the development of many cases of chronic degenerative or atheromatous valvular disease. In most of these cases, however, "when we have prescribed that continual temperance in all things which is necessary to the care of most disorders, how often do we find that our patient reckons the loss of pain to be purchased too dear by the loss of pleasure." (Gee.)

The chronic valvular disease being a fact, the treatment varies with its stage. During the period of compensation, the one important desideratum is so to regulate the patient's life that his activities may be well within the potentialities of his myocardium. This may be estimated by determining the relative amount of hypertrophy and of dilatation, the reaction of the heart and blood vessels to a measured amount of work (such as walking, climbing stairs, lifting weights, etc.), and by the condition of the other organs (passive congestion, etc.). In most cases it is wise to explain to the patient the condition of his heart and blood vessels; to tell him that although disabled (impaired), his heart is still capable of many years of effective work, provided the demands made upon it are well within its reserve power, but that this reserve power is less than in normal subjects. The patient's entire life, his diet, his work, his rest, his exercise, must be adjusted to the capabilities of his heart. The diet should be nutritious and readily digestible; the total amount at each meal should not be voluminous, fluids especially being restricted and taken between meals; a large meal, as a rule, should not be taken in the evening; strict attention should be paid to free daily evacuation of the bowels, and to free action of the skin (bathing). The patient should have an abundance of rest, and should be as much as possible in the open air; his occupation should not be too engrossing mentally nor too strenuous physically; and his exercise should be such as to afford pleasure, but not to provoke any manifestations of cardiac embarrassment. If, on the one hand, the desirable life entails continued repression of bodily spirits and intellectual ambitions, it has its compensation in prolonged life and postponement of the inevitable. It

is especially important to bear constantly in mind that a cardiac murmur is no indication whatever for the administration of cardiac tonics (such as digitalis); and that therapeutically a murmur is the least important of the physical signs. Furthermore, many symptoms, such as attacks of precordial distress, palpitation of the heart, dyspnœa, etc., are due to causes that excite these symptoms in non-cardiac subjects, such as dietetic indiscretions, the abuse of alcohol, tea, coffee, or tobacco, excessive venery, worry, anxiety, hill climbing, etc.; and they are to be combated by eradication of their cause, rather than by the immediate resort to digitalis and similar drugs. In some cases subjective complaints of precordial distress and palpitation are found to be associated with an excited overhypertrophied heart; the use of digitalis only adds to the patient's discomfort, which may be materially lessened by minute doses of aconite, small doses of the bromides, valerian, regulation (lessening) of the diet, and free purgation. When the disease has advanced and the compensation tends to become somewhat unstable (lessened reserve power), beneficial results may attend the judicious combination of rest, graduated exercises, hydrotherapy, small doses of digitalis or strophanthus for short periods, and free purgation. In other cases the imminence of decompensation is suggested by anemia and general failure of health, and it often yields to iron, quinine, arsenic, strychnine, etc.

Finally, in some cases suddenly, in most cases gradually, more or less complete failure of compensation ensues; the indications to be met are to reduce the work demanded of the heart and to increase the strength and vigor of its contractions. The most important is, temporarily at least, to reduce the work demanded of the heart—which is best effected by eradication of the cause (excessive mental or physical work, worry, anxiety, excitement, dietetic and other indiscretions, malnutrition, etc.) sometimes directly responsible for the breakdown, by rest in bed, regulation of the diet, and depletion of the engorged venous system. The rest in bed should be absolute; nothing else is so important or so efficacious. Rest in bed for ten days or two weeks of itself often suffices to restore compensation so seriously disturbed as to lead to considerable œdema; with recurring breaks in compensation rest in bed may continue to suffice, in which event digitalis and other cardiac tonics need not be given; they should be reserved until needed—which assuredly tends to prolong the patient's life. The diet should be carefully regulated to suit each individual patient, the main indications being to nourish the patient (concentrated, readily digestible, solid and semisolid foods), to prevent flatulence (avoidance of food likely to undergo fermentation), and to limit the amount of fluid intake (which not only overloads the blood and adds to the work of the heart, but also distends the stomach and mechanically adds to the embarrassment of the heart). The digestive processes may be much improved by free purgation by blue mass (or calomel) and salines, which serve to deplete the engorged gastro-intestinal and hepatic systems.

In many cases the foregoing do not suffice, or, having once or twice

proved efficacious, they are inadequate in a later recurrence of decompensation; recourse must be had to drugs that increase the strength and vigor of the cardiac contractions. Digitalis is the sovereign remedy. Since it increases the tonicity, contractility, excitability, and rhythmicity of the cardiac muscle, it is indicated in the failure of compensation that may develop in any and all forms of valvular disease. The special indication for its use is insufficiency (dilatation) of the myocardium, manifested by cyanosis, dyspnœa, venous stasis, and œdema, rather than any special valvular defect per se. The best effects of the drug are seen in cases of dilatation with final tricuspid insufficiency, congestion of the viscera, and œdema. In appropriate cases, its good influences are obvious often within twenty-four hours: the volume of the heart lessens; the cardiac action becomes regular and vigorous; the pulse becomes full, strong, and regular; dyspnœa, cyanosis, and cardiac palpitation lessen; the urine increases in amount; and the œdema begins to be absorbed. It is no less efficacious in aortic insufficiency with dilatation and venous congestion than it is in mitral valve disease; the theoretical objection to its use in aortic insufficiency, based upon its prolonging the ventricular diastole, is more than counterbalanced by its action in promoting a full, vigorous, and efficient systole with complete emptying of the ventricle, in raising the tension in the systemic arteries, and in leading to the establishment of circulatory equilibrium in the capillaries and veins.

There are three good preparations—the powdered leaves, the infusion, and the tincture; the infusion is perhaps the best, but must always be freshly prepared from the leaves. It may be serviceably combined with alkaline diuretics:

℞—Potassium acetate,
Potassium citrate, of each . . . . . 2 drams 8|0
Infusion of digitalis . . . . . 6 ounces 200|0

M. S.—One tablespoonful (15 c.c.) every four hours (or at longer intervals, as may be required).

Failure in the use of digitalis is attributable to inefficient preparations made from inert leaves (or the preparation of a supposed infusion from a mother fluid extract or similar preparation), insufficient dosage, and unsuitable case. When the stomach is or becomes deranged (nausea, vomiting, etc.), a fat-free tincture may be employed, or the infusion may be given by the rectum; or digitalin (germanicum, $\frac{1}{12}$ to $\frac{1}{6}$ grain, 0.005 to 0.01 gram) or digalen (a soluble digitoxin) may be given hypodermically; or in suitable cases digalen or digitalone may be given intravenously. Digitalin or digalen also may be given by the mouth when a prolonged mild effect is desired; but one must remember that most of the commercial alkaloids or so-called active principles are inert.

Digitalis, however, must be employed with circumspection; it is not indicated in any case of cardiac disease without evidence of myocardial insufficiency; nor in cases of rapid, irregular, or overacting heart

unattended with the aforementioned signs of failing myocardium; nor in cases of aortic insufficiency manifested only by dyspnœa, precordial pain, anginoid attacks, etc.; nor, as a rule, in high arterial tension, and angina pectoris, except perhaps in some cases in elderly subjects with an associated valvular lesion and failing compensation, in whom it often is of much service, and in whom it may be combined with vasodilators.

As a rule, the drug should be discontinued as soon as the desired results have been obtained; it may be resorted to again should the indication arise. In cases in which its use has been continued for some time, toxic symptoms may develop; but there is little evidence of the sudden development of the much-feared so-called cumulative action. Nausea and vomiting are the earliest symptoms; these may be followed by slowing and weakening of the pulse, lessening in the amount of urine, and fall in the blood pressure. More marked evidences of intoxication consist of the pulsus alternans, the pulsus bigeminus, and the development of a type of heart block (toxic depression of conductivity and contractility).

Of the substitutes for digitalis, strophanthus (or strophanthin) is the best, but it is much less efficient; it is said to cause less vasoconstriction than digitalis, and may be used when a mild effect is desired, or after the use of digitalis for some time, or in children or elderly subjects. Excellent results sometimes attend the intravenous injection of strophanthin ($\frac{1}{130}$ gram, 0.0005 grain). Convallaria, caffeine, adonis venalis, sparteine, cactus grandiflorus, squill, etc., occasionally are useful substitutes, but they are much less efficient than digitalis, and their preparations are often inert. Strychnine, iron, arsenic, and alcohol are often very useful adjuvants to digitalis, and may be used as the patient convalesces.

Attacks of acute heart failure, which sometimes are the initial manifestations of final decompensation, but which also frequently develop during the persistence of more or less well-marked failure of compensation, often demand immediate and active treatment. Frequently the attacks are due to obvious causes (dietetic indiscretions, constipation, exertion, excitement, etc.)—which should be avoided. When developed the attacks should be met with diffusible stimulants and carminatives, such as aromatic spirit of ammonia, compound spirit of ether, brandy, whiskey, strong coffee, etc., by the mouth; or whiskey, caffeine, camphor in oil, strychnine, digitalin, strophanthin, adrenalin chloride, etc., hypodermically. In many cases the most effective treatment consists of prompt venesection and the abstraction of a pint or more of blood.

The obtrusive symptoms of decompensation are usually relieved by improvement of the condition of the heart, but sometimes they require special or additional treatment. Dyspnœa not improved by the measures already mentioned is commonly due, in part at least, to some associated condition, especially hydrothorax, chronic bronchitis, flatulency, dyspepsia, ascites, etc. Hydrothorax (usually right sided) is very common and is often overlooked. It should always be sought, and when found, paracentesis should be immediately resorted to. This is an

exceedingly important therapeutic measure, and is usually promptly followed by great relief and improvement in the condition (removal of one cause of embarrassed heart action). The dyspnœa due to flatulent dyspepsia may be controlled by regulation of the diet and the use of carminatives, such as brandy, aromatic spirit of ammonia, spirit of lavender, resorcin, charcoal, sodium bicarbonate, and bismuth. Nocturnal dyspnœa, especially the awakening with a start and subsequent dyspnœa, should be controlled with morphine hypodermically. Small doses ($\frac{1}{16}$ to $\frac{1}{12}$ grain, 0.004 to 0.005 gram) often suffice; such is the relief that in suitable cases the drug should not be withheld, but its use should not be long continued. Nitroglycerin is sometimes of service in cases with high arterial tension. Distressing palpitation is usually associated with dyspnœa, is due to similar causes, and is to be similarly treated. In addition, hot moist applications or an ice bag to the precordium are often grateful. The palpitation of excessive muscular hypertrophy is often best relieved with tincture of aconite (1 to 2 minims, 0.06 to 0.12 c.c., thrice daily). Anginoid pains are often provoked, as are attacks of palpitation and dyspnœa, and should be similarly met. The anginoid pains of aortic lesions are often much benefited by nitroglycerin and potassium iodide. Nausea and vomiting require limitation of food, free purgation (calomel and salines), and bismuth, cerium oxalate, dilute hydrocyanic acid, carbolic acid, creosote, etc., and the non-use of digitalis by the mouth. Restlessness and insomnia are often distressing and weakening to the patient. The bromides, valerian, nervines, whiskey or brandy in hot water, and hypnotics, such as chloralamid, sulphonal, veronal, paraldehyde, etc., are often more or less efficacious; but in many cases nothing is comparable to morphine (or codeine or heroin)—which in aggravated cases should not be withheld, although it must be used with circumspection and not too long continued. Persistence of oliguria after several days treatment with digitalis calls for alkaline and other diuretics (caffeine, sparteine, theobromine, etc.). An active purgation (calomel, jalap powder, etc.) is often followed by active diuresis; in other cases the so-called Addison's, Guy's, or Niemeyer's pill renders excellent service:

℞—Powdered digitalis,
Powdered squill, of each . . . . . 20 grains 1 3
Blue mass (or calomel) . . . . . 10 grains 0 65
Mix and make 20 pills.
S.—One pill four times daily.

The ascites and anarasca that do not yield to several days of digitalis therapy should be relieved by tapping the abdomen and the use of Southey's tubes or aseptic puncture of the legs. Immediate benefit often follows, and, as after tapping a hydrothorax, the heart, relieved of much embarrassment, is likely to regain its tone.

DISEASES OF THE PERICARDIUM.

PERICARDITIS.

Pericarditis may be variously classified: It may be apparently primary (rarely) or secondary (usually); acute or chronic in onset and course; circumscribed or diffuse; fibrinous, serofibrinous, purulent, or hemorrhagic as regards the character of the exudate; and tuberculous, streptococcic, staphylococcic, pneumococcic, etc., as regards the specific etiological factor. Inasmuch as an etiological classification is scarcely feasible at the bedside, the following clinical classification will be found serviceable: Acute and chronic pericarditis; of the acute, the following forms: fibrinous, serofibrinous, purulent, and hemorrhagic; and of the chronic forms: serous and adhesive.

FIBRINOUS PERICARDITIS.

(*Dry, Plastic, or Acute Adhesive Pericarditis.*)

Etiology.—Fibrinous pericarditis occurs most commonly in early adult life (the eighteenth to the thirty-fifth year), but it may be observed also in childhood and in advanced life. It is rare as a primary, common as a secondary disorder, and it results always from infection. It is found most frequently associated with acute rheumatic fever, but occurs in tuberculous, pneumococcic, pyococcic, gonococcic, and influenzal, as well as in other infections. It occurs also in chorea, chronic alcoholism, gout, diabetes, scurvy, purpura, anemic and cachetic conditions, acute and chronic nephritis (frequently as a terminal infection), etc. It is often associated with myocarditis and endocarditis, the disease in reality being a pancarditis; and it frequently results from extension of disease of adjacent structures, such as the mediastinal lmyph nodes (whence the frequency of tuberculous pericarditis), the pleuræ, the lungs, the œsophagus, the vertebræ, the peritoneum, etc. It rarely follows local traumatism.

Pathology.—The lesions may be circumscribed or diffuse. The inflamed area becomes hyperemic, swollen, lustreless, and finally granular in appearance; and a more or less abundant, thick or thin, usually slightly adherent, fibrinous exudation is poured out. In most cases, in reality the exudation is serofibrinous, that is, there is always some fluid exudation, but this may be minimal (dry pericarditis); the fibrinous exudation varies considerably in amount, from a thin semitranslucent layer to a thick encasement that may completely envelop the heart. In cases of moderate fibrinous exudation the motion of the heart gradually produces a honeycomb-like, or a bread-and-butter, appearance (cor

villosum). The exudation causes agglutination of the opposed pericardial surfaces, and, ultimately undergoing organization, may lead to the formation of permanent adhesions and pericardial thickening. The inflammatory lesions may extend a variable distance into the myocardium.

Symptoms.—As a rule, the disease is entirely latent, and is discovered during life only when searched for. There may be a little precordial pain or distress; and some fever may be present, but this is usually an antecedent condition due to the primary disorder; it may be augmented by the pericarditis. The disease is to be recognized only by the detection of the pathognomonic friction sound. This is a peculiar rubbing, grating, or creaking, to-and-fro sound, corresponding in general to the systole and diastole of the heart, but not occurring absolutely synchronously with the cardiac sounds—lagging behind them, as it were; this is due to the fact that the greatest systolic excursion of the surface of the heart and therefore of its pericardium does not occur during the closure-time, but during the expulsion-time of the systole, especially at the termination of this period (Geigel). In some cases the sounds are systolic or diastolic only; rarely they exhibit a triple rhythm. These frictions, as a rule, are best heard over the normal area of superficial cardiac dulness (where the heart is closest to the chest wall) or toward the base, near the root of the aorta; they are often sharply localized to a small area, which, as a rule, is not a "valve area," and they are not transmitted as are endocardial murmurs; they give the impression of being superficial, close to the surface; they vary in intensity and are sometimes temporarily absent; and they may be intensified by the pressure of a stethoscope, a full inspiration, the patient's leaning forward, etc.

Diagnosis.—The diagnosis rests solely upon the detection of the characteristic pericardial friction. Pleuropericardial frictions may be differentiated by their common situation at the left edge of or beyond the limits of the cardiac dulness; by their being much influenced by the phases of respiration; that is, although perhaps systolic and diastolic in time, they are often intensified during expiration and lessened or abolished at the height of full inspiration.

Prognosis.—In many cases the prognosis is that of the primary disease, the pericardial lesions being more or less inconsequential. Slight lesions may subside, and leave perhaps only a localized area of thickening of the epicardium (so-called milk-spots); usually, however, more or less obliteration of the pericardial sac ensues. If the lesions do not subside, they progress to serofibrinous pericarditis.

Treatment.—The treatment is identical with that of the early stage of serofibrinous pericarditis.

SEROFIBRINOUS PERICARDITIS.

(*Pericarditis with Effusion.*)

Etiology.—There is no sharp line of demarcation between fibrinous and serofibrinous pericarditis; in many cases they represent different stages of the one process—alike etiologically (bacteriologically) and pathologically. The cases with much serous effusion, however, are most common in young male adults, and are usually associated with rheumatism, tuberculosis, or mild pyococcic infections; severe pyococcic infections usually lead to suppurative pericarditis.

Pathology.—In the early stage the lesions are identical with those of fibrinous pericarditis. Soon, or in some cases from the beginning, a more or less abundant fluid exudate is poured out—thin, serous, yellowish, alkaline, albuminous (3 to 7 per cent.), usually at least slightly turbid, and often containing fibrinous flacculi; occasionally the fluid may have a greenish tint or it may be purulent or hemorrhagic (suppurative or hemorrhagic pericarditis). The fluid varies in amount, as a rule, from 100 to 2000 c.c. In favorable cases the inflammatory lesions subside, and are followed by whitish patches, so-called "milk spots" (evidences of past circumscribed pericarditis), or total obliteration of the sac (evidences of past diffuse pericarditis). In some cases the acute lesions become subacute and finally chronic, and lead to partial obliteration of the pericardial sac associated with a small amount of fluid. In these cases exacerbations may occur. In most, especially severe cases, the heart shows more or less marked interstitial myocarditis, and parenchymatous and perhaps fatty degeneration of the muscular fibers.

Symptoms.—Serofibrinous pericarditis, occurring in the course of many of the infectious diseases, frequently runs its course entirely unsuspected; in other cases extensive collections of fluid occasion but little distress, and are only accidentally discovered by physical examination (latent cases). In some severe cases certain well-marked though non-characteristic symptoms occur.

The general symptoms are those of the primary disease, and moderate, irregular fever—102° to 103° F. The local symptoms consist of pain, which is usually moderately distressing, but which may be severe or may be absent; and dyspnœa, in some cases progressing with the outpouring of the exudation to orthopnœa, and the other results of pressure (such as, paroxysmal cough, aphonia, pulsus parodoxus, etc.). Furthermore, the accumulating fluid, increasing the intrapericardial tension, interferes with the diastole of the heart, and the inflammatory alterations (of the myocardium as well as of the pericardium) interfere with the systole of the heart; these lead to pallor, cerebral anemia, and headache, and later cyanosis and aggravation of the dyspnœa (in part due also to compression of the lungs, especially the left), and a small, weak, and

irregular pulse. In severe cases toxic symptoms are marked—vomiting, sweats, mental hebetude, coma, delirium, etc.

Physical Signs.—Physical signs are necessary to the diagnosis. In the first stage there are the usual signs of cardiac excitement, such as excitation and frequency of the heart action, accentuated sounds, etc.; possibly a palpable friction fremitus; sometimes a little increase in the transverse cardiac dulness (from dilatation); and the pathognomonic friction sounds on auscultation. As the fluid accumulates, the apex becomes weak, and ultimately, as a rule, disappears. The maximum palpable impulse is often in the fourth or the third intercostal space; this is often erroneously interpreted as the apex beat, but, as a matter of fact, in most cases the position of the heart is not much changed; in large effusions it often descends with the descent of the diaphragm, being somewhat influenced also by the size of the heart (whether compressed or dilated). Often the apex beat can be palpated in or near its normal situation by having the patient lean forward. Especially in young subjects, as the fluid increases the pericardium may bulge and the interspaces become widened; in purulent cases there may be some œdema of the overlying skin and subcutaneous tissues.

The area of cardiac dulness becomes increased in all directions (lateral displacement of the pulmonary margins). Of especial importance is the fact that it is no longer possible to distinguish an absolute (or superficial) and a relative (or deep) cardiac dulness; the dulness is absolute throughout, and the resistance is greatly increased. The dulness varies much in shape in different cases: it is often described as irregularly triangular with a blunt apex above on a level with the second left rib or interspace, and a broad base below, extending from near the right mammillary line to beyond the left (depending upon the amount of fluid). In some cases the area of dulness is distinctly pear-shaped (or pyriform); in others the shape is that of a truncated cylinder, or trapezoidal, or is quite irregular. The shape is generally devoid of much diagnostic significance. Important signs in connection with the dulness are: disproportionate extension to the right and upward; dulness in the right fifth intercostal space extending one to two inches to the right of the sternum (Rotch's sign); an obtuse (rather than a right-angled or an acute) cardiohepatic (Ebstein's) angle; extension of the dulness to the left beyond the still perceptible apex (or heart) beat; change in the shape and extent of the dulness with change in the posture of the patient —a larger area of dulness when the patient leans forward than when he is erect or recumbent, and disproportionate displacement laterally toward the lower side when the patient is on his side; dulness near the angle of the left scapula, associated with bronchial or bronchovesicular breathing, bronchophony, and increased vocal fremitis (Bamberger's sign), which disappear or lessen when the patient leans forward (anterior prone or knee-chest posture) and breathes deeply. Similar phenomena may be encountered in the left axilla and in the right mammary region. These

signs are attributable to compression of the lung, or, as suggested by Ewart, to an altered position of the liver. The diaphragm and the left lobe of the liver are displaced downward, and may give rise to a visible and palpable bulging of the epigastrium. Compression of the left lung anteriorly may cause, in the infraclavicular region, a vesiculotympanitic note (skodaic resonance) or dulness.

In most cases, as fluid accumulates, the friction sounds lessen and may entirely disappear (especially in large effusions); often, however, they merely become more restricted in area, being still audible at or toward the base of the heart. In the absence of valvular disease the heart sounds are intact, although in the early stages they are more or less obscured by the frictions, and they become progressively less loud (muffled) as the fluid accumulates, until they may become quite faint and distant. As the fluid becomes absorbed, there is a gradual disappearance of the abnormal and a gradual return of the normal physical signs.

Diagnosis.—The condition is frequently overlooked, because of its practically symptomless course; many cases are latent, and others are fleeting, developing and disappearing with great rapidity, especially in the terminal stages of divers disorders. The diagnosis must be based wholly upon the physical signs. Less than 100 c.c. of fluid is scarcely susceptible of recognition. The nature of the fluid cannot be determined by the physical signs, but in rheumatic and tuberculous cases it is usually serofibrinous, but it may be purulent in tuberculosis; it is usually purulent in pyococcic infections; and hemorrhagic in some tuberculous cases, and in purpura, scurvy, leukemia, and anemic and cachectic conditions. It is often difficult to distinguish between pericardial effusion and dilated heart, but in dilated heart there is usually a visible wavy pulsation over much of the precordium; the superficial and the deep cardiac dulness may be differentiated; the dulness is more globular in shape, and rarely reaches as high along the right edge of the sternum, or as far out into the fifth intercostal space, and if it does there is usually a visible and a palpable impulse; Ebstein's cardiohepatic angle remains acute, or at most is a right angle; and the heart sounds are usually clear, sharp, and valvular, and a gallop rhythm may be audible. Difficulty is sometimes experienced in differentiating pleural effusion, but careful physical exploration should enable one to make the proper diagnosis.

Prognosis.—The prognosis depends upon the nature of the causative factors. Most cases due to rheumatism or tuberculosis recover after ten days or two weeks with adhesions (chronic pericarditis). In cases of severe infection, with severe general symptoms, a large amount of fluid, associated valvular disease or chronic nephritis, the prognosis is anxious; such cases often terminate fatally. Some cases become subacute and chronic.

Treatment.—In all cases, even the mildest, the patient must be confined to bed, and he should be given a light, liquid and semisolid, nutritious diet. In general the treatment must be based upon the primary

disorder (rheumatism, tuberculosis, nephritis, leukemia, etc.), if this can be determined. In many, especially rheumatic cases, prompt subsidence of the phenomena follows the administration of the salicylates (as recommended in rheumatism). The pain and discomfort of the early stages not thus relieved may be mitigated by the local application of cold (dry or wet), cupping, the use of leeches, or by a hypodermic injection of morphine and atropine; or one may use the opium and quinine pill recommended for acute pleuritis. A blister is believed by some observers to prevent or limit the effusion. When once formed, the fluid, as a rule, tends to become absorbed spontaneously. Absorption may be hastened by counterirritation (blisters or iodine locally), a light dry diet (very little fluid), a salt-poor diet, diuresis (infusion of digitalis and potassium acetate and citrate), and catharsis (concentrated saline cathartics before breakfast). Signs of embarrassed cardiac action (frequent heart beat, pulsus paradoxus, low blood pressure, pulmonary and general venous engorgement) call for withdrawal of the fluid—by paracentesis in serofibrinous effusion, by incision and drainage in purulent effusion. For paracentesis the puncture may be made in the fifth interspace in or outside the midclavicular line, depending upon the distention of the pericardium, in the fifth or fourth intercostal space along the left edge of the sternum, or in the costoxyphoid angle (directing the trocar upward and backward). Should serious cardiac weakness develop at any time, stimulants, such as alcohol, camphor, ether, digitalis, strophanthus, etc., should be resorted to, and withdrawal of the fluid should not be delayed.

PURULENT PERICARDITIS.

(*Suppurative Pericarditis; Empyema of the Pericardium.*)

Etiology.—Suppurative pericarditis is a secondary disorder that sometimes occurs in the course of various pyococcic infections (pneumonia, pleural empyema, general septicopyemia), tuberculosis, etc. It may begin as a serofibrinous pericarditis, or it may be suppurative from the beginning.

Pathology.—In cases in which the fluid exudate is rather thin (seropurulent), the changes are quite like those of serofibrinous pericarditis, but intensified; the fluid is more opaque and contains more cells, the pericardium is thickened and covered with a thick fibrinopurulent exudate, and there is a marked polynuclear leukocytic infiltration of the pericardium. In the frankly purulent cases the fluid is thicker, distinctly purulent. The lesions, as a rule, extend a variable distance into the myocardium, which may become the seat of disseminated purulent foci; the lesions may even extend to and involve the endocardium. The exudate rarely becomes in part absorbed, inspissated, and calcified.

Symptoms.—In some cases, the onset and the course of the disease for some time are identical with those of serofibrinous pericarditis, of

which the suppurative process may be an advanced stage. In other cases, following antecedent symptoms of the primary disorder, the development of the pericarditis may be abrupt, with a chill, high temperature, pain, or distress in the precordial region, and general evidences of toxemia, notably delirium (peculiar to pericarditis). In many cases the purulent character of the exudate is not suspected for some time, unless the disease develops in a frank case of septicopyemia. Continuance of fever otherwise explained, however, suggests the purulent character of the exudate, as do also progressive pallor, weakness, loss of flesh, and paroxysms of chills (or chilliness), fever, and sweats, and polynuclear leukocytosis. The physical signs are identical with those of serofibrinous pericarditis; that is, the physical signs do not disclose the character of the exudate. Œdema of the overlying skin and subcutaneous tissues is quite significant of suppuration.

Diagnosis.—The diagnosis depends upon the physical signs of fluid in the pericardium, œdema of the overlying skin, general and local evidences of septicopyemia, and the results of exploratory puncture; this should not be neglected nor long postponed in doubtful cases.

Prognosis.—The prognosis is bad, as a rule, on account of the process itself and of the usually pyococcic infections of which it is a part.

Treatment.—The treatment is essentially surgical—incision and drainage, which should be instituted as soon as the diagnosis is made; and, as stated, attempts should be made to recognize the condition as soon as possible by resort to exploratory puncture. Otherwise the treatment is that of pyococcic infections in general.

CHRONIC PERICARDITIS.

Chronic pericarditis occurs in three forms:

1. **White Patches or "Milk Spots."**—These are usually found on the anterior surface of the visceral pericardium. Usually they are evidences of previous localized pericarditis, although they have been attributed to a low grade of chronic hyperplasia of the connective tissue, the consequence of the mechanical irritation occasioned by the continued impact of the heart against the resisting chest wall. Commonly insusceptibile of diagnosis, the condition is of no clinical importance, except in that it may be the source of frictions, sometimes interpreted as endocardial murmurs in apparently healthy persons.

2. **Chronic Pericarditis with Effusion.**—The exudate is usually serofibrinous, sometimes hemorrhagic in character, and not infrequently there are some soft adhesions or partial obliteration of the sac. Tubercles may be detected by macroscopic and microscopic examination. Sometimes an apparently primary disorder, it is usually part and parcel of a more or less widespread tuberculosis. The disease is suggested by general and gradual failure of health, subacute onset, chronic course, "tuberculous habitus," the constitutional symptoms of tuberculosis,

tuberculosis in the antecedents, tuberculous foci in other parts of the body, and the physical signs of pericardial exudation, its persistence, and the physical signs of fluid in other serous cavities. In the absence of clear evidence of tuberculosis elsewhere in the body, diagnostic help may be found in tuberculin injections, and in the injection of the fluid exudate into guinea-pigs. The prognosis is unfavorable. The treatment is that of tuberculosis in general, and of serofibrinous pericarditis.

3. **Adhesive Pericarditis (Obliterative Pericarditis; Adherent Pericardium; Concretio Cordis cum Pericardio; External Pericarditis; Pleuropericarditis; Indurative Mediastinopericarditis).**—Inasmuch as chronic obliterative pericarditis, external pericarditis, pleuropericarditis, and indurative mediastinopericarditis in many cases represent but stages of the same disease, and can scarcely be differentiated the one from the other, it is the part of wisdom and utility to discuss them together.

Etiology.—Adhesion of the two layers of the pericardium may be the consequence of a previous acute pericarditis, but the absence of symptoms in some cases leads to the inference that the lesions were chronic from the beginning. It is sometimes tuberculous in nature; and occurs in rheumatic, syphilitic, and nephritic subjects.

Pathology.—The two layers of the pericardium are usually intimately and completely adherent, and in many cases also much thickened, in consequence of hyperplasia of the connective tissue and the organization of a fibrous exudate. At times the adhesions being incomplete, the space thus left may contain more or less serofibrinous or inspissated and caseous material. More or less extensive calcification is not very uncommon. Associated with obliteration of the pericardial sac (internal pericarditis) there is frequently external pericarditis and indurative mediastinitis (adhesion of the pericardium and the heart to the chest wall, the pleuræ, lungs, and tissues of the mediastinum); in some cases also obliteration and extensive thickening of the pleuræ; and in other cases adhesions to the under surface of the diaphragm (perihepatitis, perisplenitis, and deforming peritonitis—multiple serositis) may be observed. If the adhesions are slight or moderate the heart may show little or no change for a long time. Usually, however, inasmuch as the adhesions are dense and extensive, there is more or less extensive hypertrophy of the heart and fibrous myocarditis, which in time are succeeded by dilatation.

Symptoms.—Clinically three classes of cases may be distinguished: (1) Cases in which the pericarditis runs its course entirely devoid of symptoms, and constitutes merely an accidental finding at necropsy; (2) cases in which, sooner or later, symptoms of disturbed or impeded cardiac action manifest themselves, and in which from the physical signs the obliterative pericarditis is more or less susceptible of diagnosis; and (3) cases characterized clinically by marked ascites, with little or no œdema of the legs, cases in which the diagnosis of cirrhosis of the liver is usually made, and which at the necropsy reveal chronic obliterative

pericarditis and certain morbid changes in other tissues and organs—notably pleuritis, peritonitis, perihepatitis, nutmeg liver, red atrophy of the liver, cirrhosis of the liver, etc.

In the majority of instances when chronic adhesive or obliterative pericarditis gives rise to symptoms, the symptoms that arise are those of failing cardiac compensation—palpitation of the heart, dyspnœa, cough, cyanosis, generalized œdema, effusions into the several serous cavities of the body, etc. On the contrary, in the cases that simulate cirrhosis of the liver, clinically, the first symptoms are sensations of fulness, oppression, and weight in the upper abdomen, or there may be apparently a sudden onset with acute pain; the abdomen increases in size and may attain very large dimensions—due evidently to ascites that may necessitate repeated tappings (Rumpf's patient was tapped 301 times). With increase in the amount of the ascites the abdominal walls become tense and painful, and the other subjective symptoms become markedly aggravated. There may be slight and transitory jaundice. Later, œdema of the legs may develop. In a few cases œdema of the legs comes on early; it may remain but a short time, disappear, and not recur until shortly before the death of the patient. In rare cases it persists; but it is always inconspicuous when contrasted with the excessive and constantly recurring ascites, and it scarcely attracts attention.

Physical Signs.—Physical signs are essential to the diagnosis. Of these the most important are: A weak or absent apex beat, especially significant in cases in which there is no increase n the area of cardiac dulness; systolic retraction of a considerable area about the apex-beat; systolic retraction of the base of the left chest posteriorly, in the region of the eleventh or twelfth rib (Broadbent's sign), attributable to adhesion of the heart, pericardium, and diaphragm, and a systolic tug upon the diaphragm which becomes communicated to its point of attachment to the chest wall posteriorly; arrest of the normal respiratory movements in the epigastric angle; a systolic tug in the epigastrium or in the neighborhood of the left seventh or eighth costal cartilage; imperfect descent of the apex beat during inspiration; inadequate or entire absence of shifting of the apex beat, with change in the posture (lateral posture) of the patient; absence of change in the limits of cardiac dulness during the respiratory phases; absence of increase of the cardiac dulness to the right, despite marked engorgement of the veins of the neck; a diastolic shock or rebound of the heart and precordium; evidences of dilatation or hypertrophy of the heart (which may be marked) in the absence of valvular or other disease that might cause it; absence of the characteristic changes in the heart in the presence of definite valvular disease—that is, absence of the usual hypertrophy of the right ventricle in mitral valve disease, and of the left ventricle in aortic valve disease; absence of pericardial effusion in the presence of pleural and peritoneal effusions; paradoxic pulse—inspiratory diminution in the force and volume of the arterial pulse; diastolic collapse of the veins of the neck (Friedreich's sign); inspiratory swelling of the veins of the neck (Kussmaul's sign);

a presystolic murmur (Fisher's sign) in the absence of other evidences of mitral stenosis or of aortic insufficiency (Flint murmur); and, as suggested by Musser, the non-occurrence in young subjects with valvular disease and cardiac insufficiency of the characteristic improvement following the administration of digitalis. None of these signs is pathognomonic of adherent pericardium; all will not be present in every case, but in many cases a sufficient number will be present to warrant the diagnosis.

Prognosis.—From the nature of the disorder, restoration to the normal is impossible. The outlook depends upon the establishment and maintenance of an efficient hypertrophy of the heart.

Treatment.—The treatment is that of myocardial insufficiency and cardiac decompensation in general. Brauer has suggested loosening the adhesions surgically (cardiolysis), an operation perhaps permissible in young subjects and in others in whom the myocardium is in good condition.

HYDROPERICARDIUM.

(*Dropsy of the Pericardium; Pericardial Transudate.*)

Hydropericardium, the transudation of non-inflammatory fluid into the pericardial sac, is a secondary process that occurs in conditions which lead to œdema generally, such as chronic heart disease with decompensation, as well as chronic disease of the lungs that induce myocardial disorders; it occurs also in the advanced stages of chronic nephritis, and in anemia and cachectic states generally (carcinoma, sarcoma, pernicious anemia, leukemia, scurvy, etc.), in consequence of hydremia and perhaps diseases of the capillary vessel, and toxemia. The *symptoms* are scarcely suggestive, and, as a rule, are overshadowed by those of the primary disorder; dyspnœa, cyanosis, and other evidences of cardiac embarrassment, are usually marked. Hydropericardium is suggested by the primary causative factors, the physical signs of fluid in the pericardium, the absence of friction sounds and of fever, and the presence of fluid in the other serous sacs. The *prognosis* is bad. The *treatment* is that of the primary cardiac, renal, or other disorder, to which, should the symptoms be urgent, paracentesis of the pericardium should be added.

HEMOPERICARDIUM.

(*Blood in the Pericardium.*)

In many cases of serofibrinous pericarditis the fluid exudate contains a variable number of erythrocytes, which, however, usually pass unnoticed, unless the fluid is examined microscopicly. Sometimes the erythrocytes are present in such numbers as to give a more or less well-marked hemorrhagic tint to the fluid. This occurs, especially in certain cases of tuberculosis, attended by the eruption of miliary tubercles

in the pericardium (rupture of new-formed capillaries); in primary and secondary tumors of the pericardium; in asthenic and cachectic states, such as chronic nephritis, purpura, scurvy, leukemia, etc. More obvious blood in the pericardium may result from perforating ulcers of adjacent organs (œsophagus, etc.), and it may be due to rupture of the heart or of an aneurysm of the ascending aorta. In the first-mentioned cases the *symptoms* and physical signs are those of serofibrinous pericarditis with exudation; in the last mentioned, those of sudden heart failure, usually soon succeeded by death.

PNEUMOPERICARDIUM; PYOPNEUMOPERICARDIUM.

(*Air and Gas in the Pericardium.*)

Pneumopericardium or pyopneumopericardium is a rare condition that may result from trauma (or wounds); from perforation from an adjacent air-containing organ, such as the lung, bronchus, œsophagus, stomach, or intestines; or from infection by Bacillus aërogenes capsulatus. Infection by Bacillus aërogenes capsulatus usually occurs shortly before death, and the lesions are those of a pyopneumopericarditis without clear cause. Most cases result from perforation of an adjacent air-containing viscus, such as rupture into the pericardium of a pulmonary cavity, abscess, or gangrenous focus, or pyopneumothorax, or of an esophageal carcinoma, or of a gastric ulcer or gastric carcinoma. In addition to the gas the pericardium may also contain a fluid transudate, which in the great majority of cases is purulent. In rare and slightly advanced cases it may be only serofibrinous.

Symptoms.—The symptoms are those of pericarditis. The diagnosis rests entirely upon the result of the physical examination. In the recumbent posture the precordium is tympanitic, partly or entirely, depending upon whether there is little or much fluid. In most cases the note changes when the patient assumes the upright posture—the tympany low down giving way to dulness occasioned by the fluid exudate, or the heart itself. The dulness is still more noticeable in the knee-chest posture. The characteristic physical sign is a peculiar metallic splashing sound, caused by the heart churning up the fluid and gas; it is often very loud, and may be heard at a distance from the patient. A somewhat similar quality, however, may be given to the heart sounds by a distended stomach, a stomach drawn up, or pushed up, or by a pneumothorax.

The prognosis is hopeless except in a few traumatic cases, in which appropriate surgical treatment, incision, and drainage are instituted early.

CHYLOPERICARDIUM.

Chylopericardium has been observed in filariasis.

DISEASES OF THE ARTERIES.

ACUTE AORTITIS.

Acute aortitis is a rare and ill-defined disorder that is said to occur in certain infections, especially those due to the pyococci (so-called malignant endocarditis, puerperal infection, etc.), but also in typhoid fever, pneumonia, influenza, tuberculosis, etc. The lesions are analogous to those of acute endocarditis. Acute lesions may be superadded to chronic, arteriosclerotic, or atheromatous changes. The peripheral arteries also may be involved, and it is not unlikely that minor acute lesions, occurring perhaps not uncommonly in acute infections, are the starting point of the more chronic lesions of later life. There are no unequivocal *symptoms*, although substernal distress and tenderness, palpitation of the heart, dyspnœa, anginoid pain, and the physical signs of dilatation of the arch of the aorta (without concomitant signs of aneurysm) are said to be significant of acute aortitis. There may be some fever, and signs of arterial embolism. The *diagnosis* must be always in doubt. The *treatment* is that of acute endocarditis.

ARTERIOSCLEROSIS.

(*Arterio-capillary Fibrosis; Atheroma; Angiosclerosis; Atherosclerosis; Chronic Deforming Endarteritis.*)

Arteriosclerosis is a term used to denote the series of changes, largely degenerative in nature, to which the arteries are subject, and of which the conspicuous feature is fibrosis.

Etiology.—Arteriosclerosis is a normal manifestation of advancing age, being found in varying degrees after the fortieth year. It is much more common, and present in higher grade, in men than in women; the onset in women is commonly later in life than in men. The chief causes of presenile arteriosclerosis are syphilis, chronic alcoholism, chronic nephritis, plumbism, gout, diabetes, excessive muscular work, excessive eating and drinking, excessive and prolonged mental work and worry, etc. This presenile arteriosclerosis also is much more common in men than in women, largely on account of the etiological factors. The disease is rare in children, and when occurring in them seems to be related, if not to the ordinary causes, to infections, such as typhoid fever, scarlatina, influenza, etc. This may be the starting point of an arteriosclerosis that becomes more or less obtrusive in later life.

Pathology.—Adami points out that in the aorta and its main branches three forms of arteriosclerosis may be distinguished: (1) Nodose arteriosclerosis, which shows itself in the slightest cases, as a thickening and sclerosis, more particularly at or around the origins of the branches

of the aorta, as yellowish-white thickenings notably affecting the origins of the intercostal arteries. Later these grow in size and some of them coalesce, forming in the opened aorta projecting flattened nodes; these may be scattered somewhat irregularly through the aorta, tending to be more abundant and more advanced in the abdominal region. This is the form especially that passes into atheromatous degeneration and softening, the breaking down of the affected region into a granular debris—so-called atheromatous abscess; calcification may occur and lead to the formation of a so-called atheromatous plaque. (2) Senile arteriosclerosis, which is characterized by the presence of hardened and at times pipe-stem radial and other arteries; but in the aorta there may be a complete absence of nodose thickening of the intima. In its place there is a diffuse dilatation of the aorta, affecting more particularly the thoracic portion; the aortic walls are thinned, instead of thickened, and the increased volume of the aorta tends to show itself not only in breadth, but also in length, the vessel being somewhat tortuous; the arch takes a wider sweep, the abdominal section is curved. In the common iliac and the carotid arteries there are slight depressions, tending to have their long axes situated transversely—the reverse of the sclerotic nodosities, a giving way taking the place of thickening of the wall. Although there may be a diffuse thickening of the intima of moderate grade in these cases, it is not the cause of the hardening of the arteries, this being due to calcification of the media (Klotz), which is likely to be preceded by marked hypertrophy of the middle coat. This Adami regards as the uncomplicated type of senile arteriosclerosis (Monckeberg's type), but it is relatively uncommon; the commonest condition in elderly people being a combination of the two types mentioned. (3) Syphilitic arteriosclerosis, which has its seat of election in the ascending aorta and the arch, where the nodes are likely to lie in groups. These in their earlier stage are large, succulent, and semi-translucent or hyaline in appearance; they have little tendency to atheromatous or calcareous change, but exhibit a later scarring or central depression with some puckering. Adami further states that the primary disturbance in these cases is a subacute mesaortitis, with small-celled infiltration around branches of the vasavasorum and absorption of the elements proper of the media. Coincidently there is overgrowth of the intimal tissue, and when as a result the deeper portions of the overgrowth exhibit necrotic change and degeneration, the underlying inflammatory granulation tissue advances new capillaries into the necrotic area. The result is an absorption of the degenerated material, replacement by cicatricial tissue, shrinkage, and scarring. There is a lack of this vascularization and replacement in the ordinary nodose arteriosclerosis. Adami speaks of the smaller arteries as—the muscular arteries and the arterioles: the muscular arteries exhibit changes similar to those mentioned in the three types of sclerosis, but there is less atheromatous and degenerative change; the calcification that does occur in the middle-sized arteries involves the media more than the intima; and there is

more or less diffuse thickening of the intima and overgrowth of the intima, suggesting a change to compensate for thinning of the media; the arterioles exhibit in general similar changes, but in one series of cases hypertrophy of the muscular coat is the dominating change, in another series fibrosis is the most evident alteration—fibrosis of the intima, fibrosis replacing largely the muscular tissue of the media, and fibrosis and pronounced overdevelopment of the adventitia.

Adami describes the following as the effects of syphilis on the aorta, and states that the lesions of the other forms of arteriosclerosis are quite similar: The primary disturbance is a granulomatous inflammatory degeneration of the media, which leads to a local giving way of the media (fatty degeneration, necrosis, and calcareous infiltration of the muscular fibers, and loss of elasticity of the elastica). If this giving way be moderate, it results in a strain hypertrophy of the intima and the adventitia, with the development of nodose intimal sclerosis; if it be extreme, overstrain, atrophy of the intima, and aneurysmal formation ensue. The intimal nodosities are not of inflammatory type and are non-vascular, although with the progressive laying down of layer upon layer of connective tissue on the more internal aspect of the intima the earlier and deeper placed layers of new tissue gain less and less nourishment, and are likely to exhibit fatty degeneration and necrosis. These products of necrosis exert a chemotactic influence upon the nearby vessels of the medial granulation tissue, with, as a result, a secondary and later entrance of new vessels into the early and deeply placed atheromatous area, absorption of the necrotic products, replacement by granulation tissue, contraction of the granulation tissue, and depression and scarring of the sclerotic nodules so characteristic of syphilitic sclerosis. Strain and overstrain are believed to be of the greatest importance. As regards the significance of heightened blood pressure (hyperpiesis, Allbutt), Adami reaches the conclusion that like results may ensue when, on the one hand, the artery has undergone weakening and the blood pressure is normal, and when, on the other hand, the artery has no preliminary degeneration of its media and the blood pressure is above the normal. The increased blood pressure may be variously provoked. By some observers it has been attributed to hyperactivity of the adrenals, perhaps induced by divers toxic causes.

Symptoms.—The symptoms, like the lesions, as a rule, are insidious in onset and slow in progression, although rarely they seem to develop with considerable celerity. Even considerable degrees of arteriosclerosis, however, may be present for years without occasioning notable symptoms, and symptoms when they do occur are most diverse in nature, due to the fact that although the arteriosclerotic process may be widespread, the lesions are often unequally pronounced in different parts of the body.

In the early stages there is lessened elasticity and contractility of the vessels and consequent imperfect and irregular distribution of the blood—which may be a more or less permanent condition or only induced by

exertion, fatigue, etc., and the ensuing symptoms are correspondingly variable. In general they are those of lessened vigor and vitality, of fatigue, neurasthenia, etc. The patient readily becomes tired, even more or less exhausted; he is nervous and irritable, unable to concentrate his attention as formally, and suffers from headache, vertigo, insomnia, or somnolence; exertion may provoke various painful sensations, such as headache, paresthesias, neuralgiform pains, or muscular cramps; the functional activity of the special senses (especially hearing and sight) becomes impaired; and various other symptoms develop, depending upon the special prominence of the lesions in the different organs.

Those referable to the cardiovascular system naturally are usually in the ascendency. The heart passes through all the stages from efficient hypertrophy to final dilatation and decompensation. More or less dyspnœa and palpitation of the heart are usually present on exertion; epistaxis and other hemorrhages are not uncommon; later, œdema and other signs of cardiac failure supervene. In general appearance the patient may be ruddy, full-blooded, or he may be pale and anemic looking—which is due to imperfect distribution of the blood, and not to poor blood, at least in the beginning. In the early stages major diagnostic importance attaches to visible and palpable evidence of thickening and tortuosity of the superficial arteries, to increased blood pressure (160 to 180 mm. Hg. or more), to the physical signs of hypertrophy of the left ventricle, a dull, prolonged, somewhat muffled first sound at the apex of the heart (augmented muscular element), an accentuated aortic second sound, and a sloping ascent, broad apex, and slow decline of the pulse wave, especially apparent in tracings. Ultimately the common signs of dilatation ensue; indeed, these may be the symptoms presented by the patient when he first comes under observation. Involvement of the coronary arteries may lead to angina pectoris, the fibroid heart, aneurysm of the heart, thrombosis with sudden death, etc.

Renal symptoms are often quite conspicuous—those of chronic diffuse indurative (interstitial) nephritis; indeed, in most cases, the disorder is correctly described as cardiovascular-renal disease. Symptoms referable to the nervous system are often prominent; of these, the most important are headache, vertigo, tinnitus aurium, incapacity for work, and irritability; transitory attacks of hemiplegia, monoplegia, or aphasia (sometimes attributed to arterial spasm and consequent temporary anemia); cerebral hemorrhage (often the immediate cause of the fatal issue).

Gastro-intestinal symptoms—that is, so-called indigestion, but also some cases of gastric ulcer, are sometimes attributed to special involvement of the supplying vessels. Special interest attaches to attacks of severe abdominal pain (abdominal angina) that come on several hours after eating, and may be associated with other anginoid phenomena, such as angina pectoris; they are attributed to spasm of the gastric and mesenteric vessels. Severe painful cramps in the legs

induced by walking are not infrequent (intermittent claudication or lameness); the disorder is due to temporary anemia of the affected parts, and is characterized by the absence of symptoms when at rest, by their provocation by walking, by their cessation upon resting, and often by the absence of a palpable pulsation in the pedal arteries.

Obstruction to arteries elsewhere may give rise also to local pain, and if the obstruction become complete gangrene may follow.

Diagnosis.—The diagnosis may be suggested by the known habits of the patient, his general appearance (full-blooded or anemic looking), and gradual impairment of vigor and vitality; confirmation of the diagnosis is found in the obvious condition of the peripheral arteries, the hypertrophy of the left ventricle, an accentuated aortic second sound, and the pulse. In most cases also the blood pressure will be found high, but in many quite advanced cases, the blood pressure is low, perhaps normal or less. All early diagnoses may sometimes be made by ophthalmoscopic examination of the retinal vessels.

Prognosis.—The outlook in general is unfavorable, but it should not be exaggerated, since the disorder is one evidence of advancing age, Occurring in young subjects, however, arteriosclerosis usually shortens life, and is dangerous on account of the possibility of apoplexy, uremic manifestations, heart failure, etc. In many cases the progress of the disease may be stayed by appropriate treatment.

Treatment.—The disorder can be more readily prevented than cured. All the known causative factors should be avoided, especially excessive eating and drinking, alcohol, tobacco, tea and coffee, excessive mental and bodily work, etc., and the patient should be instructed to lead a quiet life free from unnecessary cares and excesses of all kinds. Diseases known to lead to arteriosclerosis should be efficiently treated, especially syphilis, gout, diabetes, lead poisoning, chronic nephritis, etc. In many even non-syphilitic cases the iodides seem to do good, and may be given (30 grains, 2 grams, daily) for a month at a time and then discontinued for a month. Excessive blood pressure should be reduced by nitroglycerin (not longer than three or four weeks at a time), sodium nitrite, erythrol tetra-nitrate, free catharsis, a low diet, and rest; but one must remember that some of the increased pressure is a conservative process, and an attempt to reduce the pressure to normal should not be made. Much benefit often attends a course of treatment at one of the well-known spas, or a course of massage, passive exercise, and warm baths. When the heart begins to fail, the treatment is that of the fibroid heart.

ANGINA PECTORIS.

(*Stenocardia; Heart-pang; Breast-pang.*)

Angina pectoris is a disorder usually associated with sclerosis of the first portion of the aorta and the coronary arteries, and characterized by paroxysmal attacks of excruciating pain in the region of the heart,

a sensation of constriction of the chest, and a feeling or fear of impending death, and sometimes sudden death.

Etiology.—Angina pectoris is a disease of adult life (after the fortieth year) and of the male sex; women are very rarely affected. The etiological factors are those of arteriosclerosis, especially alcoholism, syphilis, chronic nephritis, gout, excessive muscular and mental work, etc.

Pathology.—The lesions found in fatal cases consist of sclerosis and atheroma of the first part of the aorta, of the coronary vessels (especially their mouths), and of degenerative (fibroid and fatty) changes of the myocardium. Often there is associated disease of the aortic valve and orifice, or aneurysm of the aorta. In some cases degenerative changes have been found in the cardiac ganglia. Rarely the disorder is found in association with mitral disease. In a few cases no noteworthy lesions have been found (perhaps they have been overlooked).

The nature of the disease has not been fully elucidated. It is assuredly absurd to describe it as a neurosis, as is commonly done. Clifford Allbutt, with much reason, regards the disorder as a painful lesion of the first part of the arch of the aorta: an aortitis, rheumatic or influenzal, etc. (in which cases the tensile stresses may be about or even below normal), or atheroma of the aorta (in which cases the tensile stresses may be normal or excessive). He believes that sometimes the disorder is produced by stretching of the pericardium, as in aneurysm of the left ventricle, or by inflammation of that part of the tunic which invests the root of the aorta, and that very rarely it may depend on some extraordinary kind of disturbance in the mitral area. Mackenzie points out that the symptoms occur in patients with the most diverse forms of lesions, even in the absence of any evidence of cardiac disease: the most common conditions are aortic aneurysm, aortic valvular disease, atheroma of the coronary arteries, myocardial degeneration or enfeeblement from poor nourishment or overexertion, and increased arterial pressure usually associated with coronary sclerosis and myocardial degeneration in elderly subjects. He further points out that in these conditions angina pectoris is a late symptom, developing after the heart has been struggling for a long time against obstacles opposed to an efficient action, or after the nutrition of the muscle has been impaired by gross pathological changes in the coronary artery, or when the myocardium has become impaired through slowly advancing degeneration of the muscle fibers; that many patients when at rest do not suffer from pain, but that any cause inducing increased work in the heart provokes an attack. He concludes that angina pectoris is an evidence of exhaustion of contractility, and occurs when the contraction meets a greater resistance than it can easily and efficiently overcome, whether a fairly strong muscle struggles against an increased resistance, or when a weak or degenerated muscle has opposed to its contraction a normal or even lowered pressure, but a pressure greater than the weakened muscle can readily overcome. Huchard attributes the attack to anemia of the heart muscle the result

of disease or spasm of the coronary arteries, and compares it to intermittent lameness or claudication.

Symptoms.—The chief manifestation of angina pectoris is paroxysmal pain; but excluding the rare cases of sudden embolic plugging of the coronary artery, this paroxysmal pain occurs only after a long period of gradual enfeeblement of the myocardium, and it is almost always induced by some extra demand upon the heart, such as muscular effort, mental excitement, increased peripheral resistance (chilling of the surface, etc.), flatulence, etc. The paroxysms may vary considerably in severity, from slight precordial pain or constriction of the chest to the most severe attacks. These consist of paroxysms of excruciating pain in the region of the heart, a sensation of constriction of the chest, especially in front, and a feeling of exhaustion and mental anguish amounting to an apprehension of impending death. The pain is usually of the most excruciating character; it irradiates from the cardiac plexus behind the aorta along the distribution of the first four thoracic and the seventh and eighth cervical nerves, that is, to the left shoulder, arm, and neck; rarely also to the right side, or the abdomen. The associated sensation of tightness or constriction of the front of the chest is attributed by Mackenzie to spasm of the intercostal muscles induced by reflex stimulation. Phenomena of vasomotor disturbance are usually present, such as pallor (occasionally flushing) of the face, profuse perspiration, etc. The patient may be absolutely immobile, with a staring countenance, completely conscious, but unable to articulate; or he may be restless and anxious; in either event he is usually overcome emotionally by the greatest mental anguish. During the attack the heart is variable; often it reveals no changes aside from the phenomena of arteriosclerosis, being quite normal in rate, rhythm and strength; but the pulse may become soft, compressible, and scarcely perceptible (weak heart), or extrasystoles and the alternating pulse may occur. Mackenzie states that the blood pressure may not be increased. Dyspnœa is not a phenomenon of angina pectoris per se, although it may be induced by associated conditions. The attack lasts from a few seconds to a minute or thereabouts; rarely, however, severe pain or a succession of attacks may continue for an hour or more. The paroxysm may lead to immediate death (even the first paroxysm), or it may subside as suddenly as it began, or at its height the patient may pass into syncope. The end of the paroxysm may be associated with the eructation of gas (unconsciously sucked in during the attack), the passage of a large amount of urine, etc. After the attack the patient feels exhausted for several hours or several days, and some substernal distress may persist for a short time.

Recurrences may ensue for several days in succession with increasing weakness of the myocardium; or at irregular, more or less long, intervals, over a period of years; or the first may prove to be the only attack. Cessation of the attacks may follow dilatation of the heart and relative mitral insufficiency (Musser), but the ordinary signs of decompensation are likely soon to supervene.

Diagnosis.—The severe paroxysms are sufficiently characteristic as scarcely to be mistaken. Slight attacks are often overlooked or misinterpreted, but it is significant if they are provoked by exertion, excitement, etc., and if they are associated with anginoid attacks in the legs (intermittent lameness), or in the gastro-intestinal tract (abdominal angina) etc. The so-called pseudo-angina that occurs in hysterical, neurasthenic, and other nervous disorders must be excluded. Huchard points out that it is most common in women of all ages; the attacks are spontaneous, and often periodical and nocturnal; the pain is less severe, lasts a long time, and is associated with other nervous phenomena—vasomotor and neuralgiform; it is not fatal; and it is improved by medication directed to the nervous system. A toxic angina is sometimes set up by tobacco, coffee, etc. Nothnagel described a vasomotor angina, characterized by coldness and numbness (vasomotor spasm) of the extremities, faintness, and precordial pain.

Prognosis.—The condition, of course, is grave, but not so apprehensively so as is often maintained; it depends upon the efficiency of the heart muscle, and the care with which the patient regulates his life. While the first attack may be, it is usually not, fatal, and proper treatment may lead to cessation of the attacks.

Treatment.—The treatment in general is that of arteriosclerosis and the fibroid heart, the chief indications being to reduce the pressure against which the more or less exhausted myocardium must contend, or the stress or tension to which the sore aorta (Allbutt) is subjected, and to avoid all the factors that may provoke an attack. Underlying conditions such as rheumatism, gout, syphilis, diabetes, flatulence, etc. must receive appropriate treatment. The patient must bring his daily work well within the capabilities of his heart; he must avoid all overexertion, and lead a quiet life. A prolonged holiday or a course of treatment at some one of the well-known spas is often very efficacious. In some cases rest in bed for a time is essential. The diet must be restricted and carefully regulated, an excess of meats and especially of carbohydrates being avoided, especially at the evening meal; and cardiac excitements, such as alcohol, coffee, tea, tobacco, must be prohibited. The frequent use of mercurial and saline laxatives is very valuable. The iodides seem to be of unquestionable service; potassium, sodium, or ammonium iodide should be given in small (3 to 5 grain, 0.2 to 0.3 gram) doses for a long time, with short periods of intermission (one week in four); or one may use nitroglycerin for the physiological effect, in frequently repeated doses (since the effect of each dose does not last more than an hour); or one may give erythrol tetranitrate ($\frac{1}{2}$ grain, 0.03 gram, three or four times daily), or the nitrites—which have a more lasting effect.

The paroxysms are best treated with amyl nitrite, of which 3 to 5 minims (0.2 to 0.3 gram) should be given by inhalation. A subject of angina pectoris should carry glass capsules or perles containing the mentioned dose, and immediately upon the onset of the paroxysm break one

in his handkerchief and inhale it; relief is usually prompt and complete. In some cases benefit does not ensue, in which event resort should be had to morphine ($\frac{1}{4}$ grain, 0.015 gram) and atropine ($\frac{1}{100}$ grain, 0.0006 gram) hypodermicly; or, as recommended by Osler, a few whiffs of chloroform (it must be used with caution, since it may prove a dangerous remedy). Recurring nocturnal attacks may be sometimes prevented by chloral, the bromides, valerian, etc. Allbutt recommends the continuous use of atropine, in increasing doses as tolerance is established, with a view to blocking the impulse by which the heart is inhibited. The treatment of the eventual failure of the heart is that of the fibroid heart; digitalis, however, as a rule, and similar cardiac tonics are distinctly contra-indicated.

ANEURYSM.

An aneurysm is a circumscribed tumor containing fluid or solid blood, communicating directly with the canal of an artery, and limited by the tunic which is called the sac (Hilton Fagge).

Etiology.—The causative factors in general are those of arteriosclerosis, of which aneurysm is a result. Major significance attaches to syphilis and excessive muscular strain, whence aneurysms are most common in the male sex (86 per cent. of the cases). They develop most commonly between the thirtieth and the fortieth year, that is, they "coincide with that time of life when the period of greatest bodily vigor overlaps the beginnings of the period of the occurrence of atheroma" (Coats).

Pathology.—True and false aneurysms may be distinguished, the true aneurysm being one in which the aneurysmal sac consists in whole or in part of the wall of the bloodvessel, the false being one in which the aneurysmal sac consists of connective tissue, the wall of the bloodvessel having ruptured and permitted the extravasation of blood into the surrounding connective tissue; but one must bear in mind that the sacs of many so-called true aneurysms in their later stages, at least, are composed largely of connective tissue, the walls of the bloodvessel originally constituting the sac having become atrophic, degenerated, and replaced by newly formed connective tissue. A true aneurysm may be, and usually is, circumscribed and saccular, that is, it communicates with the lumen of the vessel by a narrow opening called the neck; or it may be ectatic, that is, merely a dilatation of the vessel, and fusiform, cylindrical, or cirsoid in shape; or it may be dissecting, that is, the result of rupture of the intima and consequent infiltration of the blood between the coats of the vessel, whence it may find its way into the lumen of the vessel again or outward into the adjacent connective tissues. Quoting Coats, an aneurysm may be said to be the result of "a local disproportion in the normal balance between the amount of lateral pressure inside the artery and the elastic resistance of the vascular walls, arising either from abnormal increase of the former or diminution of the latter; most frequently, indeed, from both these causes

combined." The increased pressure is a concomitant and causative factor of the arteriosclerotic process; the diminution in the elastic resistance of the vessel is also part of the arteriosclerotic process, but the local predisposition that determines the site of the aneurysm is a congenital inherent localized weakness of the vessel wall, or an acquired deficiency of vitality that may be only a special localization of the sclerotic process or the result perhaps of some infection passed through in early life.

Saccular aneurysms may be single or double (rarely multiple); and very small, scarcely more than a slight bulging or pouching of the vessel, or they may attain the size of a child's head. Seventy-five per cent. of all aneurysms involve the thoracic aorta, and of these, 59 per cent. the ascending portion of the arch, 29 per cent. the transverse portion of the arch, and 12 per cent. the descending portion (Crisp).

Rarely other types of aneurysms occur, such as: embolic and mycotic aneurysms due to mechanical or infected emboli (they are usually multiple); a traction aneurysm on the under surface of the aortic arch, at the site of the insertion of the remnant of the ductus Botalli; an arteriovenous aneurysm, in which a communication is established between an artery and a vein; should the communication be direct the condition is spoken of as an aneurysmal varix; should a sac intervene, it is termed a varicose aneurysm.

Symptoms.—An aneurysm may be for a long time latent; indeed, the first manifestations may be the fatal rupture and hemorrhage. In most cases, however, sooner or later, manifestations develop, in the one case subjective, the aneurysm of symptoms; in the other case objective, the aneurysm of physical signs. The symptoms are all pressure effects. Pain is most significant; although absent in some cases, it may be the most obtrusive manifestation of the disease. It is most likely to be present in deep-seated aneurysms, and is usually described as sharp and paroxysmal, or dull and boring. Persistent pain beneath the sternum, or in the region of the fourth, fifth, or sixth thoracic vertebræ, is extremely significant, especially if it be unrelieved by medication. Pressure on the intercostal nerves may give rise to the most intractable neuralgiform pain, which may be referred to the front of the chest, and be for some time the only symptom.

In a majority of the cases the most noteworthy pressure signs are connected with the respiratory tract, of which the most important are a characteristic cough, alterations in the voice, and dyspnœa, due to paralysis or spasm of one or both vocal cords, occasioned by pressure on the recurrent laryngeal nerve or nerves, generally the left. The cough is brassy, metallic, clanging, or ringing in character—a laryngeal cough, and of itself often permits a presumptive diagnosis. The alterations in the voice have been well described by Walshe, who says that "the speaking voice may be husky, muffled, cracked, and hoarse, or simply weakened, or tremulous and variable in pitch, or actually lowered in register." In some cases complete

aphonia results. Laryngoscopic examination frequently reveals the larynx normal except in one respect—the cadaveric position of the vocal cord corresponding to the recurrent laryngeal that is paralyzed. In these cases the other vocal cord crosses the median line and renders phonation possible, but in consequence of unequal tension, unequal vibrations, and hoarseness, or a peculiar monotone and inability to reach a high note, a falsetto voice develops. Dyspnœa, which is often associated with marked stridor that may be inspiratory, or inspiratory and expiratory, and which like the cough is often paroxysmal, is an extremely characteristic sign. Sometimes the narrow glottis is still sufficient for easy respiration during quiescence, but exertion produces increased respiratory demand, and during the effort the paralyzed cords are violently drawn downward and inward, with resulting apnœa. In some cases the trachea, or more commonly one of the bronchi, usually the left, becomes compressed, and gives rise to paroxysms of dyspnœa, to lessening of the respiratory murmur over a part or the whole of the corresponding lung, and perhaps also other signs of compression and atelectasis of the lung (diminished expansion, dulness on percussion, indefinite signs of solidification, etc.). If the pressure be long continued, retention of secretion, fetid bronchitis, and bronchiectasis may result; and pressure on the bronchial arteries may lead to gangrene of the lung or indirectly to tuberculosis (a not uncommon complication). Aside from large and often fatal hemorrhages, small hemorrhages (so-called weeping of the aneurysm) may occur, being due to rupture of inflammatory capillaries in the trachea or bronchus at the point of compression, or to leakage through the sac that has ulcerated into the trachea or bronchus or lung. The trachea may be displaced laterally as well as anteroposteriorly, and the normal up and down movements during deglutition may be restricted. Tracheal tugging may be elicited—a distinct tug or downward traction on the trachea occurring synchronously with the systole of the heart (Oliver's sign); a lateral rather than a downward traction is sometimes apparent (Cardarelli's sign). Both are most common in aneurysms of the transverse or descending arch of the aorta that impinge on the bifurcation of the trachea or the left bronchus. A tracheal diastolic shock also may sometimes be detected (Hall's sign).

Pressure on the œsophagus occasions dysphagia; but it is remarkable that in some cases of even large aneurysms the œsophagus escapes. Dysphagia may also be due to pressure on the vagus or its œsophageal branches, in which event there may be also other manifestations of gastro-intestinal disorder—vomiting and diarrhœa, but these are unusual. The dysphagia is due to an œsophageal stricture (which it virtually amounts to); and it is important to bear in mind that in the event of suspected œsophageal stricture a bougie or stomach tube should not be passed until the possibility of aneurysm has been excluded; on more than one occasion a stomach tube has been inadvertently introduced into an unsuspected aneurysm. Pressure on the thoracic duct may occur, and lead to emaciation; whence it may be difficult to determine whether

emaciation is due to such pressure or to œsophageal obstruction and the ingestion of insufficient food.

The aneurysm may be so situated as to interfere with the blood supply to one arm or one side of the neck and head. At times this results from direct pressure on the supplying artery; in other cases it results from the artery springing from the aneurysmal sac, the blood supply of the part in question being compromised by the blood being obliged to pass through a sac more or less filled with blood clot. Examination of the corresponding arteries of the two sides of the arm, head, and neck may disclose retardation and weakness or lessened volume of the pulse on the affected side; but one must bear in mind anatomical differences in corresponding arteries. As pointed out by Osler, in some cases of large saccular aneurysm the normal pulse in the abdominal aorta and its branches (femorals, etc.) may be obliterated. At times, although scarcely as frequently as in intrathoracic tumor, obstruction to the venous circulation occurs—congestion and swelling of the veins, cyanosis, and sometimes œdema, corresponding in situation and extent to the situation and degree of the venous obstruction. In some cases more or less well-marked evidences of a vicarious collateral circulation appear in the normally small veins beneath the skin. Clubbing of the finger ends and incurving of the nails, usually unilateral and corresponding with that toward which the pressure of the aneurysmal sac is directed, have been observed in some cases.

Pressure on the ciliospinal branches of the sympathetic nerve is by no means uncommon. Should it be moderate, sufficient only to irritate or stimulate the vasodilator fibers, dilatation of the pupil and pallor of the corresponding half of the face result; should the pressure be sufficient to paralyze or destroy the vasodilator fibers, the pupil becomes contracted, and the half of the face hyperemic and the seat of increased perspiration. In some cases there may be narrowing of the palpebral fissure and enophthalmos. Exophthalmos has been observed; it has been attributed to retrobulbar venous engorgement, but it is inexplicable in some cases (perhaps stimulation of the unstriped orbital muscle attached to the eye ball).

Physical Signs.—The important physical signs consist of a visible and palpable abnormal expansile pulsation, usually to one side of or beneath the sternum, or in the episternal notch, or in the left back or axilla. Over the pulsation a thrill, usually systolic, occasionally also diastolic, may be palpable; a diastolic shock is common, and is due sometimes to augmented recoil of the aortic leaflets (when the valve is competent), sometimes to recoil of the aneurysmal sac or the aorta between the sac and the aortic valve. The aneurysm occasions an area of dulness which usually merges with the cardiac dulness; this dulness is of the greater significance the farther it extends from beneath to either side of the sternum in the first, second, or third intercostal space. Dulness may be found also posteriorly, usually beneath or to the left of the vertebræ. In deep-seated aneurysms no dulness may be elicited; and in other cases the dulness may be obscured by co-existing emphysema.

Auscultation may reveal no abnormalities. In the majority of cases, in the early stages at least, two sounds are audible over the aneurysm —the two sounds heard normally in the aortic area. Some observers attribute the second sound to the aneurysm itself, which perhaps may be the case in associated aortic insufficiency, in which, however, a second sound may not be heard either at the aortic area or over the aneurysm. In some cases a systolic murmur is audible over the sac; in other cases a double, systolic and diastolic, murmur; the diastolic murmur may be produced in the aneurysm or it may be due to a commonly associated aortic insufficiency. The murmur may be audible in the trachea or in the open mouth of the patient. The heart is, as a rule, more or less displaced, and commonly reveals the physical signs of more or less hypertrophy, which is due to the associated arteriosclerosis, aortic valve disease, etc.; the aneurysm as such produces little if any hypertrophy.

Diagnosis.—The diagnosis depends in part upon the symptoms, and in part upon the physical signs. The association of an otherwise inexplicable cough, dyspnœa, and interthoracic pain should always awaken the suspicion of deep-seated aneurysm. Percussion and palpation of a localized area of the anterior chest may provoke paroxysms of severe pain and coughing, which Eichhorst believes significant of aneurysm. Aneurysms of the ascending aorta usually grow from the anterior surface forward and to the right, and present in the first, second, or third right intercostal space, and produce, as a rule, obtrusive physical signs. Aneurysms of the transverse arch usually grow from the concavity of the arch downward and backward; symptoms rather than physical signs are likely to be obtrusive, although dulness may be found beneath and to the left of the sternum, or in the back. Aneurysms of the descending arch are likely to grow from the posterior surface, and to be for a long time latent or to produce indefinite or ill-defined symptoms. In all cases of aneurysm, the most conclusive diagnostic aid is furnished by the use of the *x*-rays.

In the differential diagnosis one must exclude other forms of pulsation, such as the so-called dynamic pulsation, which is common in the aorta in cases of aortic insufficiency, and in the abdominal aorta; even widespread and forcible pulsations of this sort often subside remarkably under the influence of rest. The palpable pulsation of the abdominal aorta common in thin and neurotic individuals is all too commonly interpreted as an abdominal aneurysm. The diagnosis of an aneurysm of the abdominal aorta depends upon the detection of a definite palpable expansile tumor. Pulsating empema in the neighborhood of the heart should not occasion any diagnostic difficulty. Most difficulty is experienced in distinguishing between an aneurysm of the aorta and a solid *mediastinal tumor;* but aneurysm is most common in males, between the ages of thirty-five and fifty-five, especially those who have been engaged in laborious occupations, who have become infected with syphilis, and who have used alcohol to excess; intrathoracic growths exhibit no special predilection for either sex; they occur at any age

if secondary, but usually before the age of thirty-five if primary, and occupation, alcohol, and syphilis (except in cases of gumma) are without etiological significance. A bulging expansile tumor is pathognomonic of aneurysm; in the case of intrathoracic tumor, a primary growth elsewhere in the body or the development of a mass of enlarged lymph nodes above the clavicle is likewise practically pathognomonic. Œdema of one side of the chest, especially if unassociated with œdema of the corresponding arm, is suggestive of tumor. A systolic or a double murmur, if unassociated with other evidence of valvular disease, is suggestive of aneurysm; murmurs, as such, have no relation to intrathoracic growths, but they may develop if the heart be displaced or the bloodvessels be encroached upon or distorted. A palpable diastolic shock and a marked accentuation of the aortic second sound is quite common in aneurysm, but usually absent in tumor. Tracheal tugging does not occur in tumor, although a slight systolic movement of the trachea (observable even in some healthy persons) may be detected. The effects of aneurysm on the lung are usually those of compression only, whereas the effects of tumor growth may be manifest by solidification, the growth invading the lung tissue and in some cases giving rise to metastatic nodules. In the case of tumor a clear, or probably more likely a blood-stained, fluid is common when the growth has reached and invaded the pleura; should an aneurysm give rise to alterations in the pleura, acute pleuritis usually results. Pain is usually less severe, and is certainly less characteristic in tumor formation than it is in aneurysm, although it may be entirely absent in some cases of aneurysm. Pressure effects on the recurrent laryngeal nerve are more suggestive of aneurysm than of tumor. Under the influence of rest, potassium iodide, and other treatment, the symptoms of aneurysm usually, for a time at least, show amelioration, whereas almost without exception the symptoms of tumor are continuously progressive. The course of aneurysm is very variable and it may last for years, whereas intrathoracic growths almost certainly result fatally within a year from the onset of symptoms.

Prognosis.—The outlook in aneurysm is bad, since from the nature of the disease the outcome is necessarily fatal; occasionally a spontaneous cure results. The usual duration of life after the diagnosis has been made is from two to three years, although it may be prolonged by appropriate treatment. Death usually (75 per cent. of the cases) results from rupture and hemorrhage into the pleura, the pericardium, the trachea, a bronchus, the œsophagus, or externally after erosion and perforation of the sternum and the adjacent costal cartilages or ribs. Death may be due also to asphyxia from compression of the trachea or a main bronchus, to inanition from compression of the œsophagus of the thoracic duct, to embolism especially of the brain, etc.

Treatment.—The main object of treatment is to promote clotting within the sac and consequent cicatrization. These are best achieved by rest and a restricted diet (so-called Tufnell treatment), which reduce

the force and frequency of the heart beat. The rest should be absolute in bed for two or three months, and should be combined with mental quietude and freedom from excitement and worry. The diet should be reduced to a minimum: Tufnell advised only ten ounces of solids and eight ounces of fluids—a regimen which is scarcely necessary, and if rigidly insisted upon usually induces the patient to seek another physician. After the early rest in bed, if the patient has submitted to it, he should be directed to lead an orderly and quiet life, the danger that threatens, on excitement, exertion, and augmented blood pressure being pointed out to him. His bowels should be moved freely daily with laxatives, so as to avoid the straining inevitable in constipation. The iodides (30 to 60 grains, 2 to 4 grams, daily), continued for a long time, unquestionably do good in many cases, the syphilitic as well as the non-syphilitic; usually they notably mitigate the pain, and in some cases they seem to promote clotting within the sac. Calcium lactate (40 to 60 grains, 2 to 4 grams, daily) may be given for four or five days from time to time with the same object in view. Gelatin is thought also to promote clotting, and may be given with the food, but its value, aside from the contained calcium, is doubtful. Pain may be somewhat relieved by hot or cold applications, counterirritation, etc., but it is sometimes so severe as to demand morphine, which from the nature of the disease need not be withheld from time to time, although it should be used with circumspection. Aconite is sometimes of service in quieting an overactive heart. The introduction of gold wire and electrolysis to promote rapid clotting has been successfully performed in a number of cases, with relief to the patient's suffering and prolongation of his life. Obviously it should be employed only in sacculated aneurysms.

SECTION VII.

DISEASES OF THE RESPIRATORY SYSTEM.

Pathological Physiology.—The respiratory system is designed to subserve the function of gaseous exchange between the body and its environment; that is, the absorption by the blood circulating in the pulmonary capillaries of oxygen from the air contained within the pulmonary alveoli, and the giving up by the blood to the air of carbon dioxide. This, the so-called external respiration, is much related to and upon it depends the so-called internal respiration—that is, the gaseous exchange that takes place between the blood and the tissues, the absorption by the tissues of oxygen essential to their vital, or metabolic, activities, and the giving off into the blood of carbon dioxide, one of the end-products of katabolism. The lungs, therefore, are primarily concerned in the oxidative processes, of which the metabolic and nutritive processes largely consist; and the life history of the oxygen taken into the body by way of the lungs (to a limited extent also by the skin) soon becomes associated with that of the food taken in by way of the digestive system; but this is a subject that pertains more to disturbances of nutrition than of respiration.

The lungs are prevented from collapsing by the pressure of the atmosphere (15 pounds to the square inch) which reaches the lungs by way of the air passages and cannot exert a compressing force on the lungs from without, on account of the resistance of the bony and muscular framework of the thorax. In conditions of deficient entrance of air into the lungs considerable depression of the intercostal spaces may occur (especially noticeable in children with laryngeal or tracheal obstruction); and should the bony framework of the chest become softened (as in rickets) notable deformity may and usually does ensue. In the event of perforation of the chest wall and the consequent access of the air to the pleural sac, the lung collapses, usually to about one-fifth its normal volume; this is due not only to the atmospheric pressure as such, but also to overcoming of the normal negative intrapleural pressure (4 to 6 mm. Hg.), and to the activity of the contractility of the lungs. The contractility of the lungs is the resultant of the large amount of elastic tissue in the pulmonary parenchyma and of the musculature of the bronchi; these, when normal, give the lungs their normal tone and are the basis upon

which normal pulmonary percussion resonance depends. This contractility of the lungs, as well as the negative intrapleural pressure that it occasions, is of much importance in disease; it may become impaired from overstrain by increased intrapulmonary pressure (blowing wind instruments, etc.) and by disease of the elastica, as happens in emphysema, as well as by compression of the lung by a collection of fluid in the pleura, or extensive disease (such as pneumonia) of one lobe; in all of these conditions the percussion note becomes vesiculotympanitic in character. The contractility of the lung on the non-diseased side also is a factor in causing displacement of the heart and mediastinum in conditions, such as pleural effusion or transudation and pneumonothorax, in which the one lung becomes more or less collapsed; and the negative intrapleural pressure is doubtless a factor in inducing hydrothorax when the pleural vessels become passively congested (chronic cardiac and pulmonary disease).

Both phases of respiration, inspiration and expiration, result from muscular activity, notably of the scaleni and intercostal muscles and the diaphragm; both are due to the rhythmically recurring activity and relaxation of the opposing sets of muscles. While expiration is almost unopposed, during inspiration the contractility of the lungs, the elastic resistance of the chest wall, and the force of gravity (represented largely in the bony framework of the thorax) must be overcome. How irrational, then, in diseased states to add to the embarrassment of an already embarrassed respiration by the use of heavy bedclothes, a heavy poultice to the front of the chest, etc. During inspiration the lungs expand downward, forward, and laterally, the greatest excursion occurring downward and laterally, whence the friction sound of pleuritis is usually best heard in the anterolateral aspect of the chest. Comparatively little expansion occurs upward or backward; such expansion upward as does occur is due largely to descent of the lungs as a whole, a fact in part, at least, answerable for the common localization of tuberculosis at the apices.

The normal vital capacity of the lungs, that is, the greatest volume of air that can be expired after the fullest possible inspiration, varies between 3500 and 4000 c.c., averaging in the adult male about 3700 c.c. (Howell). It varies somewhat with the size of the chest, the age and height of the individual (being greater in tall than in short subjects); it is less in the prone than in the sitting or upright posture (whence the exaggerated dyspnœa of the prone posture). It diminishes in old age, in part on account of ossification of the costal cartilages and rigidity of the thorax, and it is reduced in diseases of the lungs, especially tuberculosis.

The tidal air, the air breathed in and out during ordinary quiet respiration, averages about 500 c.c.; the complemental air, the amount, in addition to the tidal air, that can be breathed in by the fullest possible inspiration, averages about 1600 c.c.; and the supplemental air, the amount which by a forced expiration can be breathed out after an ordinary quiet expiration, averages also about 1600 c.c. The tidal air, the complemental air, and the supplemental air together make up the

vital capacity. The residual air, the amount of air that remains in the lungs after a forced expiration, averages about 1000 c.c.; it becomes much increased in emphysema. In health and during normal breathing the reserve supply of air amounts to about 2600 c.c., that is the supplemental air plus the residual air. Of the 500 c.c. of air regularly inspired during quiet breathing, about 360 c.c. (Howell) reaches the pulmonary alveoli by a process of diffusion; this bears a relation to the reserve supply of air about as 1 to 7, so that one may assume a renewal of the air contained within the lungs once during each seven or eight respiratory acts.

The tidal air suffices for the ordinary requirements of life, but the increased demand for oxygen entailed by muscular exercise is met by deep breaths (supplemental air) and increased frequency of breathing. Similarly in diseased states, when the available breathing surface of the lungs becomes lessened or the blood surcharged with carbon dioxide, deep and frequent respirations (dyspnœa) are obvious efforts on the part of the body to supply a sufficiency of oxygen; this is not always successful, so that to the dyspnœa, cyanosis is added. In a healthy man weighing 70 kilograms (150 pounds) the average daily intake of oxygen is about 700 grams, and the average daily output of carbon dioxide is about 800 grams. This may be increased by excessive muscular activity and other factors.

The composition of the inspired air averages, in volume per cent., nitrogen, 79; oxygen, 20.96; and carbon dioxide, 0.04; and the expired air, nitrogen, 79; oxygen, 16.02, and carbon dioxide, 4.38 (Howell). This gain of 4.34 volumes per hundred of carbon dioxide and loss of 4.94 volumes of oxygen does not accurately represent the change in the composition of the air in the alveoli, since during ordinary quiet respiraation only 72 per cent. of the tidal air reaches the alveoli. In the alveoli the carbon dioxide reaches 5 to 6 volumes per cent., and the oxygen falls to 13 to 14 per cent. Furthermore, the air as expired has been warmed almost, if not quite, to the body temperature, whereby the body loses daily from 50 to 100 calories; and the expired air is also almost saturated with aqueous vapor, from 250 to 500 grams of water being thus lost, and in consequence of the evaporation entailing a daily loss of from 145 to 290 calories. In addition, the expired air is often said to contain a poisonous, volatile, organic substance to which the ill effects (headache, depression, etc.) of the vitiated atmosphere of badly ventilated rooms, churches, schools, and other places where large numbers of people congregate, are attributed. The results of investigations of this subject are not entirely conclusive, but there is no definite proof of the existence of such a poisonous organic substance. The unquestionable ill effects of bad ventilation are with more propriety attributed to decrease of oxygen, increase of carbon dioxide, augmented temperature of the air and its saturation with aqueous vapor, and exhalations from unclean bodies, perspiration, excretions from the sebaceous glands, and odors from clothes.

The normal body is able to withstand the effects of considerable

variations in atmospheric pressure without ill consequences; but this varies much in different individuals, and is considerably reduced in diseases of the lungs and the heart and bloodvessels. Notable reduction in atmospheric pressure, such as obtains at high altitudes (mountain climbing, balloon and aëroplane ascents, etc.), often results in so-called mountain sickness (weakness, faintness, syncope, epistaxis, etc.); and the results of increase in atmospheric pressure are well exemplified in caisson disease. Compressed air, however, may be resorted to with benefit in many cases of diseases of the lungs.

The chief results of disease of the respiratory system, in so far as this system itself is directly concerned, consist of diminution of the external gaseous exchange. This is, in part, a passive, physical process; in part, an active vital process, the result of a specific functional activity of the alveolar epithelium—which may become deranged or abolished in disease. Speaking generally, this diminished gaseous exchange may be due to impeded entrance of air into the lungs or to inability on the part of the lungs to utilize an adequate supply of air. In either event the obtrusive clinical manifestations consist of: (1) Disturbances in the rate and rhythm of respiration, and (2) of cyanosis; but both of these phenomena may be due also to disorder of the heart, as well as to other factors.

Disturbances in the Rate and Rhythm of Respiration comprise:

1. *Dyspnœa.* The term dyspnœa is generally applied to any noteworthy increase in the force, rate, or depth of the respirations; sometimes it is restricted to labored breathing, being thus distinguished from merely quickened breathing (hyperpnœa, polypnœa, tachypnœa), a condition that may be observed after muscular exercise, during nervous excitement or perturbation, in cerebral disorders, in hysteria, etc. Dyspnœa is a provision of nature designed to subserve the function of breathing, that is, the proper oxygenation of the blood, and it is the consequence of obstacles to the ready performance of this function or of increased demands for oxygenation (deficiency of oxygen or increase of carbon dioxide, which is a direct stimulant to the respiratory centre). It varies in degree in different cases—from slight breathlessness on exertion or excitement to orthopnœa even when the patient is at rest in bed. It may be objective or subjective, or both; that is, it may or may not be accompanied by subjective sensations of substernal or thoracic distress or actual pain, oppressed breathing, lack of air, and more or less anxiety and apprehension; in some cases the subjective sensations (so-called air-hunger) may entirely overshadow the objective dyspnœa. Although increased frequency of the respirations is usually an obtrusive feature, increased respirations are not always dyspnœic, and notable dyspnœa may be associated with oligopnœa. Dyspnœa may occur in the following conditions: (*a*) All painful affections of the respiratory tract (including the pleura), the thoracic wall, the diaphragm, etc. The respirations are likely to be superficial and of increased frequency. (*b*) Obstruction of

the upper air passages: obstructions within the lumen, disease of the passages themselves constricting the lumen, or compression from without. The respirations are likely to be deep, they are often infrequent, there is usually an associated inspiratory stridor, and the expiration, as a rule, is not prolonged. (*c*) Obstruction of the lower air passages—the smaller bronchi and bronchioles, such as occurs in the several types of bronchitis, in bronchial asthma, etc. The respirations are likely to be increased in frequency, and there is usually prolonged expiration and expiratory stridor. (*d*) Disorders of the pulmonary alveoli, in which the available breathing surface becomes restricted, such as pulmonary congestion and œdema (of which unstable or non-compensated cardiac disease is usually the primary factor), emphysema, inflammatory conditions (pneumonia, bronchopneumonia, abscess, etc.), as well as conditions that lead to compression of the lungs (pleural and pericardial collections of fluid, pneumothorax, mediastinal tumors, distortion of the vertebræ, displacement upward of the diaphragm, etc.). The respirations are usually of increased rate, force, and depth. (*e*) Infectious febrile conditions generally. The increased respirations are due to a direct action of the bacterial toxins and the augmented temperature on the respiratory centre and to surcharging the blood with carbon dioxide, a consequence of increased metabolism. (*f*) Intoxications, such as uremia and diabetic coma, in which Kussmaul's dyspnœa, or air-hunger (deep, energetic, long-drawn inspirations contrasting with short, sighing expirations), may be observed. (*g*) Anemic conditions, in which the available hemoglobin may be so small that even frequent and deep respirations do not suffice to satisfy the demand for oxygen.

2. *Oligopnœa.* Infrequent breathing may be regular or irregular (arrhythmic). It results from depression of the respiratory centre, and may be encountered in—some severe infections; exogenous poisonings, such as opium and chloral poisoning; autogenous intoxications, such as uremic and diabetic coma; nervous disorders, such as meningitis, hemorrhage and tumor of the brain; and the death agony.

3. *Arrhythmic Breathing.* There are two chief types of peculiarly arrhythmic breathing: (*a*) Cheyne-Stokes breathing, which occurs in arteriosclerotic subjects (cardiovascular-renal disease), uremia, myocardial degeneration, conditions of increased intracranial pressure, etc., and is characterized by longer or shorter periods of apnœa (cessation of breathing) alternating with periods of breathing, at first slow and shallow, but gradually becoming deeper and more rapid until they reach a maximum, whereupon they gradually decrease and finally again cease; and (*b*) Boit's, or meningeal, breathing (which, however, occurs in conditions other than meningitis); it is characterized by more or less periodic, but sometimes irregularly recurring, pauses in breathing lasting from a few to thirty or more seconds, and is unattended by the characteristic increase and decline of the respirations of the Cheyne-Stokes breathing. The causes of the periodicity of these types of breathing are not well understood. Both are of serious prognostic significance.

4. *Asphyxia or Suffocation.* Complete obstruction of respiration and final cessation of breathing may result from: (*a*) Subjection to an inert or irrespirable atmosphere; (*b*) obstruction of the larynx or trachea by an impacted foreign body, false membrane, tumor, etc., or by compression from without, as by an aneurysm, mediastinal tumor, goitre, etc., or by hanging or strangulation; (*c*) laryngeal spasm or œdema or paralysis of the vocal cords; (*d*) drowning; (*e*) inundation of the lungs by fluid from within the body—blood (ruptured pulmonary bloodvessel or aneurysm), serous fluid (severe, especially acute, pulmonary œdema), pus (retropharyngeal abscess, etc.); (*f*) intoxications, such as accompany chronic passive congestion from decompensated cardiac disease, in which the excess of carbon dioxide poisons the respiratory centre; morphine, chloral, or chloroform, which poison the respiratory centre directly; and coal-gas poisoning, in which carbon monoxide replaces the oxygen from the hemoglobin; and (*g*) severe and prolonged tetanic convulsions (strychnine poisoning, tetanus, status epilepticus).

Cyanosis. Cyanosis commonly attends dyspnœa and is due to like causes, to a deficiency of oxygen and an excess of carbon dioxide in the blood. Dyspnœa, however, may occur without cyanosis, as in uremia, and cyanosis may be present without dyspnœa, as in congenital heart disease. Cyanosis is common in diseases of the lungs interfering with proper oxygenation of the blood, and may result from obstruction to the entrance of air into the lungs, to restriction of the available breathing surface of the lungs, and to weakness of the respiratory muscles and nerves. It occurs also in disease of the heart, in erythremia (page 385), in chronic acetanilide and other coal-tar poisonings, and in so-called enterogenous cyanosis (page 414).

There are a number of acts, such as coughing, sneezing, sighing, yawning, sobbing, hiccough, etc., that may be looked upon as modified respiratory movements. Coughing, the most important of these, is primarily and essentially protective in character, being designed to rid the upper air passages of irritating bodies or particles or fluid secretions; it may also be produced by irritating vapors, and by stimuli originating in various parts of the body and communicated to the pneumogastric nerve. The chief sources of such stimuli comprise the larynx, the posterior aspect and the bifurcation of the trachea, and the pleura. So-called gastric, hepatic, splenic, and uterine coughs are sometimes described, but there is no proof whatever of their acutal occurrence. In the event of pathological secretions being present in the air passages, coughing subserves the useful purpose of their elimination; it should not, therefore, be unwisely suppressed at all hazards, since retained secretions are likely to undergo decomposition and may bring in their wake conditions other than those to which they owe their origin. Often, however, it is excessive and disadvantageous, distending the lungs unduly in consequence of increased intrapulmonary pressure, increasing the intrathoracic pressure and impeding the flow of blood to the heart, and subjecting the patient to the danger of rupturing a bloodvessel; in these

cases the cough per se should be kept in control. On the other hand, weak or ineffectual coughing or the inability to cough may prove a source of danger to the subject, leading to retention of foreign bodies, pathological secretions, etc.; this occurs especially in aged and infirm persons, in those debilitated from any cause, in muscular asthenia, especially of the respiratory muscles, in diabetic, uremic, and other forms of coma, in anesthetization, and in grave disorders of the nervous system. The retained secretions, being usually infected, commonly lead to bronchopneumonia and often a fatal termination.

DISEASES OF THE NOSE.

EPISTAXIS.

(*Nose-bleeding.*)

Etiology.—Epistaxis, the most common mucous-membrane hemorrhage, may be due to general or local causes. The general causes are those that determine hemorrhage in other parts of the body, such as: (1) The acute infectious diseases, especially typhoid fever (being characteristic of the onset), diphtheria, scarlatina, measles, pneumonia, influenza, etc.; (2) blood dyscrasias, such as purpura, scurvy, hemophilia, leukemia, pernicious anemia, etc.; (3) conditions of increased arterial tension, such as plethora (especially after unusual exertion and suddenly increased blood pressure), arteriosclerosis (nose-bleeding of the aged), chronic interstitial nephritis, etc.; (4) chronic venous congestion—that of advanced cardiac and pulmonary disease, as well as the more local congestion produced by tumors of the neck, etc.; (5) certain poisonings (phosphorus, salicylic acid, etc.); (6) ascent to high altitudes (mountain climbing, balloon ascensions); and (7) it may occur as a manifestation of vicarious menstruation. The local causes of epistaxis are commonly traumatic—injuries to the nose (fracture of the nasal or cranial bones), the presence of foreign bodies, the violent use of instruments, excessive "nose blowing," and "nose picking" of children; and local diseases, such as acute and chronic rhinitis; diphtheria, tuberculosis, syphilis, glanders, and other ulcerative processes; benign and malignant tumors, etc. Osler has described a multiple telangiectasis of the skin and mucous membrane with recurring epistaxis.

Pathology.—The bleeding may result from capillary oozing or from rupture of the vessel wall (traumatism or ulceration). The frequency of this nasal hemorrhage is due to the abundant vascular supply and the absence of adequate support to the vessels, which conditions are most marked over the middle and inferior turbinate bones, where the veins form a well-marked venous plexus. When bleeding is the result of local ulceration, it generally comes from an area on the cartilaginous

septum about half an inch from its anterior end. This has sometimes been described as the site of predilection. In most cases the hemorrhage takes place from only one nostril.

Symptoms.—Following traumatism the hemorrhage is sudden and may persist for some time, or it may cease suddenly. When due to fracture of the anterior fossa of the base of the skull, it is usually continuous though slight, and is generally mixed with cerebrospinal fluid. When due to local causes, it may be sudden, severe, and often very difficult to control. When due to general diseases, it may take place on the slightest provocation and varies in severity. The blood may be arterial or venous. The quantity may vary from a few drops to several pounds. It is rarely dangerous to life.

Diagnosis.—The diagnosis is usually quite evident; but occasionally the source of the bleeding may not be immediately apparent. Blood from the nasal mucous membrane may be swallowed (especially during sleep), and being later vomited, may simulate true hematemesis; or it may enter the pharynx and, being expectorated, simulate true hemoptysis. Examination of the nasal mucosa and attention to the associated phenomena should prevent diagnostic errors.

Prognosis.—The prognosis depends altogether upon the cause of the bleeding and the general condition of the patient.

Treatment.—In most cases the hemorrhage ceases spontaneously. Should this not occur, the nasal cavities should be examined to determine the seat of the bleeding. Divers sorts of local applications are often effectual—adrenalin chloride, hot water, ice water, cocaine, alum, zinc, hamamelis, tannin, iron perchloride, etc. Persisting hemorrhage from a ruptured vessel may sometimes be stilled by the direct application of chromic acid, the Paquelin cautery, etc. Should these measures prove ineffectual, the nares should be plugged (anteriorly, posteriorly, or both) with cotton or similar material, or a thin rubber finger cot may be introduced into the nares and then distended with air or water. Internal remedies are of little value, except perhaps calcium lactate in certain general disorders associated with lessened coagulability of the blood.

ACUTE RHINITIS.

(Acute Coryza; Acute Nasal Catarrh; Common Cold.)

Etiology.—A "cold in the head" is really an acute infection of the upper air passages; it may be merely an acute rhinitis, or the catarrhal inflammation may extend to the pharynx and perhaps involve the entire upper respiratory tract. It may occur as an independent disorder, involving persons of all ages, especially during the changeable weather of the late fall and early spring. It is commonly attributed to exposure to cold and damp, but these, as well as divers local disorders (chronic rhinitis, nasal polyps, etc.) and general debilitating conditions, act as predisposing factors; the exciting cause is doubtless some bacterium. Micrococcus

catarrhalis is believed to be the cause of most of the cases that exhibit infectious (epidemic) characteristics; other cases seem to be due to the pneumococcus, the bacillus of Friedlander, Micrococcus paratetragenus, Bacillus septus (Bacillus coryzæ segmentosus), etc. In some cases acute coryza is merely symptomatic—of certain acute general infections, such as measles, influenza, pertussis, syphilis, etc., or drug intoxications, such as the iodides, the bromides, etc.; or it may result from the inhalation of irritating dust or vapors (acids, ammonia, etc.).

Pathology.—The nasal mucosa is swollen, congested, œdematous, and covered with a profuse, clear, alkaline, mucous secretion—which is also abundantly discharged through the nares. The lesions may extend to the pharynx and the upper respiratory tract generally, the Eustachian tubes, the accessory nasal sinuses, etc. After the lapse of several days the swelling and œdema subside somewhat, the secretion lessens and becomes mucopurulent in character, and gradually ceases entirely. Occasionally the nares may be more or less filled and obstructed by a fibrinous exudation (fibrinous rhinitis); this is usually due to the diphtheria bacillus, but may also be caused by streptococci, staphylococci, and pneumococci.

Symptoms.—The onset, as a rule, is sudden; not infrequently there is initial headache and chilliness (rarely a distinct chill), which are soon followed by sneezing, a sense of fulness in the nose, embarrassed breathing (nasal obstruction, often necessitating mouth breathing), general malaise, neuromuscular pains in the neck, back, and extremities, fever (100° to 101° F.), rapid pulse, and other fever phenomena. At first there is very little nasal discharge, but this soon sets in, and usually becomes abundant, clear, and watery. The sense of smell and of taste are usually obtunded. The inflammatory lesions may extend to the frontal sinuses, and occasion severe frontal headache (at the base of the nose); to the lacrymal duct, and lead to conjunctivitis and epiphora; to the Eustachian tube, and cause temporary deafness and tinnitus aurium; to the pharynx and induce local pain and soreness and dysphagia; to the larynx and trachea, and cause hoarseness, difficult phonation, and bronchial cough, etc. At the end of thirty-six to forty-eight hours, the swelling and congestion of the nasal mucosa begin to subside, nasal breathing becomes less embarrassed, the discharge becomes lessened in amount and mucopurulent in character, and convalescence is established.

Diagnosis.—The diagnosis, as a rule, is quite obvious. Care should be taken to avoid confounding a simple acute rhinitis with the cases symptomatic of one of the general infections, such as measles, influenza pertussis, diphtheria, etc.

Prognosis.—The disease usually runs a course of from three to seven days, and is usually free from complications. Rarely the accessory sinuses, the Eustachian tube, etc., may become seriously affected; or the condition (usually from repeated attacks) tends to become chronic.

Treatment.—In those subject to repeated attacks, local disease of the nose should receive attention. A hygenic mode of life may prevent

attacks or mitigate their severity—that is, suitable clothing during changeable weather, the avoidance of draughts and exposure to wet and damp, good ventilation in working rooms and sleeping rooms, frequent bathing, exercise, etc.

Many attacks are mild, do not occasion much distress or incapacitate the patient, and soon subside. But if the attack is at all severe, and if there is fever, the patient should go to bed for the first twenty-four hours at least. The time-honored purge (calomel or blue mass followed by a saline), 10 grains (0.6 gram) of Dover's powder, and hot whiskey and water (or hot lemonade) still find justification in frequent abortion of the attack; it is really good and effective treatment if the patient sweats; but he may be made to sweat otherwise, as, for instance, by a Turkish bath, which is often very beneficial. The "fulness in the head" and the embarrassed nasal breathing may be relieved by preliminary cleansing of the nares with an alkaline solution (Dobell's solution) and the use of adrenalin chloride (1 to 1000) or cocaine (1 per cent. in liquid albolene), or thymol and camphor (each 0.5 per cent. in liquid albolene). The adrenalin and cocaine may be followed by unpleasant after-effects (vascular relaxation and œdema); they should not be used too frequently, and assuredly not given to the patient for self medication. The inhalation of menthol sometimes lessens the local discomfort. In addition, relief may be afforded by the following, in a capsule or tablet, every two or three hours:

℞—Quinine sulphate	2 grains	0	13
Aspirin (or salol)	3 grains	0	20
Atropine sulphate	$\frac{1}{100}$ grain	0	0006
Camphor monobromate	1 grain	0	065—M.

If there is much fever, a mixture such as the following, is often of much service:

℞—Spirit of nitrous ether			
Spirit of camphor, of each	3 drams	12	00
Camphorated tincture of opium	2 drams	8	00
Solution of ammonium acetate, sufficient to make	3 ounces	100	00—M.

S.—Two teaspoonfuls (10 c.c.) every three hours.

A minute dose of tincture of aconite may be added to this mixture if the fever is high and there is much vascular throbbing. During convalescence from severe attacks the patient should be careful not to expose himself to inclemencies of the weather, and he should take a tonic.

CHRONIC RHINITIS.

(*Chronic Nasal Catarrh.*)

Etiology.—Chronic nasal catarrh may be due to repeated attacks of acute rhinitis; some cases seem to be chronic from the beginning, that is, the acute passes into the chronic form; other cases are due to specific infections, such as tuberculosis, syphilis, etc. Certain of the atrophic cases

(ozœna) are attributed to microörganisms, such as Bacillus mucosus ozœnæ, Bacillus fœtidus, etc. Anemia and conditions of general debility predispose to the disorder.

Pathology.—Simple, hypertrophic, and atrophic cases may be distinguished. In the simple cases the nasal mucosa is swollen and congested and covered with more or less thinnish mucous or mucopurulent exudate. The erectile tissue, especially of the turbinate bones and the septum, is especially susceptible to the action of irritants, including cold, and, becoming swollen, may occasion more or less nasal obstruction, embarrassed nasal breathing, and consequent mouth breathing. In the hypertrophic cases, in addition to the foregoing, there is more or less marked and permanent hypertrophy of the nasal mucosa, the formation of so-called polyps, and sometimes involvement of the turbinate bones, leading to partial or complete obstruction of the nasal passages. There is usually also more or less catarrh of the pharynx, with adenoid vegetations, and consequent mouth breathing, nasal voice, deafness (Eustachian tube involvement), flattened face, peculiar expression, and defective mentality (especially when occurring in young subjects); perhaps also asthma, bronchitis, etc. The atrophic cases perhaps sometimes develop out of the hypertrophic, but often they seem to be atrophic from the beginning. The mucosa is pale and thin, the erectile tissue is atrophic, and both in many places are replaced by ulcerating or scar tissue; the nasal passages are widened; there is a little mucopurulent secretion, which, drying, forms crusts and occasions an extremely offensive odor (ozœna).

Symptoms.—The chief symptom in the hypertrophic form is embarrassed nasal respiration, which is usually associated with a sense of fulness in the nasopharynx, and a more or less abundant mucous or mucopurulent excretion, which gives rise to the so-called "hawking" and spitting. The atrophic form is especially characterized by the fœtid odor and more or less local distress.

Diagnosis.—The diagnosis suggested by the symptoms, is to be made by rhinopharyngeal examination.

Treatment.—The treatment consists of mild antiseptic douches, such as Dobell's solution, etc., to insure cleanliness and remove secretions; the local use of iodine, potassium iodide, etc., in the hypertrophic form, with eventual removal of the enlarged turbinates, and of various antiseptics and deodorants in the atrophic forms. For details of the treatment one should refer to special treatises on rhinology.

HAY FEVER.

(Hay Asthma; June Cold; Rose Cold; Autumnal Catarrh.)

Hay fever is a disease provoked by the pollen of certain grains and plants, and is characterized by catarrhal inflammation of the conjunctiva and the upper respiratory tract, embarrassed respiration (asthma), and general depression.

Etiology.—Hay fever is most common in adult males, but it occurs in both sexes, especially in those engaged in intellectual as contrasted with manual pursuits. Individual sufferers exhibit a special susceptibility, which it seems may be inherited (familial predisposition), but which usually is an acquired general nervous instability or a local disorder (hypersensitiveness) of the nerve endings of the nasal mucosa. In this country, as well as in England and Germany, the disease begins toward the end of May and lasts five to eight weeks; and, especially in this country, it appears again about the middle of August and lasts again six or eight weeks or until the first frost (which it does not survive). Many subjects escape the first, but succumb to the second visitation. The exciting cause appears undoubtedly to be the pollen of certain grains and plants. Ragweed (ambrosia) and, to a less degree, goldenrod (salidago) are the main causes in this country; but grains such as rye, (especially in England), corn, etc., and other plants (chrysanthemums, asters, etc.) are also of etiological significance. All cases are not provoked by the one cause; the American disorder, for instance, seems to differ etiologically from the English, the German, etc. Dunbar has isolated from the pollen a toxalbumin which he believes to be the specific cause of the disease; it is pathogenic in susceptible individuals in doses of $\frac{1}{40000}$ milligram. The disease itself is believed to be an expression of hypersusceptibility to this toxin, which exerts a specific action on the terminal nerve fibers of predisposed persons, and has some influence also on the erythrocytes, which it lakes.

Pathology.—The lesions consist of catarrhal changes in the conjunctivæ and the upper respiratory tract. The asthma and general depression are attributable to the general influence of the toxin.

Symptoms.—The onset, as a rule, is abrupt, and in susceptible subjects develops about the same time each year. In many instances the attack resembles an ordinary acute coryza, in others, an attack of bronchial asthma; or the one may alternate with the other. At the onset, in most cases, to the more common manifestations of coryza, there are added considerable sneezing, obstructed nasal passages, conjunctivitis with marked itching of the eyelids and lacrymation, and severe itching sensations of the soft palate and pharynx; the nasal discharge is usually copious, thin, and watery—rarely mucopurulent. The catarrhal process may extend to the larynx, bronchi, and lungs, with accompanying cough and asthmatic seizures; these may even predominate, and not infrequently prevent sleep. Headache, malaise, exhaustion, fugitive pains, mental depression, and other general symptoms occur, and there may be gastro-intestinal disturbances. Variability in the severity of the symptoms may develop from day to day—mitigation due perhaps to staying indoors or to rain which precipitates the pollen, and augmentation due to going outdoors, especially in the sunshine which dries and permits diffusion of the pollen (which may be increased by high winds).

Diagnosis.—The disease is readily recognized from the rather sudden development annually, in the spring, or more commonly in August in

this country, of severe coryza, accompanied by conjunctival, pharyngeal, and bronchial (asthmatic) symptoms, and general depression.

Prognosis.—The disease usually lasts five to eight weeks and subsides spontaneously—the autumnal catarrh with the first frost. Rarely a person attacked in the spring suffers more or less throughout the summer and until October. Those once attacked are likely to suffer again, but spontaneous abatement of the symptoms is sometimes observed after the age of about sixty years.

Treatment.—The only certain means of relief is a sojourn, during the hay-fever season, at a high altitude in a region free from the ragweed, etc. The White Mountains, the Adirondacks, as well as certain mountainous regions of the South and West and Canada, afford complete immunity. Some subjects do well at the seashore. Dunbar has introduced a pollen antitoxin, under the name of pollantin (marketed in a dry and in a liquid form), which is to be used locally in the nose (and not injected hypodermicly); he asserts that he has employed it with excellent results or partial success in 87 per cent. of 1240 cases. Temporary relief is afforded by the local use of adrenalin chloride (1 to 1000), cocaine (whence the habit has often been acquired), menthol, morphine, etc., and by the internal use of atropine, which usually produces the greatest relief. S. Solis-Cohen recommends the local and hypodermic use of pituitary extracts (posterior lobe). Obvious diseases of the nasal passages should be treated appropriately (see special treatises); and the general neurotic basis of the subject should be improved by the avoidance of mental and bodily overwork, and the use of iron, arsenic, phosphorus, strychnine, etc.

DISEASES OF THE LARYNX.

ACUTE CATARRHAL LARYNGITIS.

Etiology.—Acute catarrhal laryngitis may occur at any age, but it is most common in adolescents and adults, especially in those debilitated from any cause. The exciting causes are infectious, toxic, mechanical, or thermic irritants. The infectious form of the disease may be primary or secondary. Most primary cases are related etiologically to exposure, or "catching cold," and they are provoked by streptococci, staphylococci, pneumococci, influenza bacilli, Micrococcus catarrhalis, etc. Secondarily, the disease may follow disease of the nose, throat, or bronchi, or it may occur as a manifestation or complication of certain infectious diseases (measles, scarlatina, influenza, typhoid fever, diphtheria, syphilis, tuberculosis, etc.). The toxic factors comprise the inhalation of irritant vapors (acids and alkalies); the excessive and prolonged use of tobacco, alcohol, etc.; gout, and

sometimes the use of drugs, such as mercury, the iodides, etc. The mechanical factors comprise traumatism, inhalation of dust, the action of foreign bodies, overuse of the voice, etc. The thermal factors comprise the action of very hot liquids, steam, etc.

Pathology.—The lesions consist of swelling and redness of the mucous membrane, especially in the aryepiglottidean folds. The mobility of the vocal cords is impaired; sometimes there is considerable œdema; and there is more or less mucous or mucopurulent secretion. Occasionally there are minute submucous hemorrhages, and rarely superficial erosion of the mucous membrane (catarrhal ulcers), especially on the cords, the anterior commissure, or the interarytenoid fold. Inspissation of the secretion gives rise to a condition called laryngitis sicca.

Symptoms.—At first there is a sensation of soreness or irritation and tickling or burning in the throat, a harsh, dry, sometimes spasmodic cough, and more or less alteration of the voice that may amount to complete aphonia. Phonation, deglutition, and inspiration (especially of cold air) are painful; dyspnea may be marked, especially in children, in whom it may be spasmodic and quite urgent (œdema). Expectoration at first is slight, but it soon becomes more abundant and mucous, or mucopurulent, occasionally blood-streaked. The general phenomena are usually slight—moderate fever for several days, headache, and malaise. Occasionally, especially in children and when the inflammatory phenomena are marked below the vocal cords, such narrowing of the lumen of the larynx may be produced that the symptoms resemble what have been ascribed to "croup"—crowing cough, stridor, and attacks of suffocation. These attacks often come on at night—due to accumulation of secretion and aspiration of the tongue.

Diagnosis.—The diagnosis is usually easy, even without laryngoscopic examination, which, however, may be required in doubtful cases. Laryngismus stridulus may be distinguished by the mode of onset, neurotic temperament, associated rickets, and the absence of fever, of coryza, of hoarseness, and of aphonia. Diphtheria may be distinguished by the more severe local and general symptoms, the presence of membrane on the tonsils or fauces, and early swelling of the cervical lymph nodes. Hysterical aphonia may be distinguished by the absence of fever and the results of the laryngoscopic examination.

Prognosis.—The prognosis in adults is good, as a rule, the disorder ending in recovery in from three or four days to a week or more. In children a fatal termination may be induced by suffocation or a consecutive bronchitis or bronchopneumonia.

Treatment.—Confinement of the room (the bed, in severe cases), rest to the larynx by the non-use of the voice, a mercurial purge, a diaphoretic febrifuge mixture (mentioned under coryza), and the inhalation of steam impregnated with menthol, compound tincture of benzoin, etc., suffice to bring about recovery in the majority of cases. Cold moist applications to the neck are often grateful to the patient. If the cough is very irritating, the throat may be sprayed with menthol,

cocaine, etc., or a Dover's powder or other sedative may be administered at night. The severe nocturnal attacks common in children are usually promptly relieved by an emetic and hot compresses to the neck.

ŒDEMATOUS LARYNGITIS.

(*Œdema of the Larynx; Œdema of the Glottis; Phlegmonous Laryngitis.*)

Etiology.—Œdematous laryngitis is peculiarly a disease of adult life, being uncommon in childhood. It may be primary or secondary. As a primary disorder it may be due to: (1) Infectious agents—whence it occurs (*a*) during the course of certain infectious diseases (smallpox, measles, scarlatina, typhoid fever, diphtheria, tuberculosis, syphilis, etc.); (*b*) as a concomitant of acute catarrhal laryngitis, laryngeal perichondritis, etc.; and (*c*) as an extension of the inflammatory œdema of Ludwig's angina, parotitis, tonsillitis, etc. (2) Toxic agents—inhalation of irritant vapors, the medicinal or other use of mercury, the iodides, etc. (3) Mechanical agents—foreign bodies impacted in the larynx. (4) Thermal agents—inhalation of steam. (5) Angioneurotic factors—angioneurotic œdema. As a secondary disorder it may occur (1) as a complication of general œdema (in heart disease, kidney disease, and lung disease), and (2) in consequence of pressure on the jugular veins (as by aneurysm of the aorta, tumors of the thyroid, mediastinum, etc.).

Pathology.—The lesions consist of an inflammatory œdema, involving especially the aryepiglottic ligaments, the epiglottis, and the false vocal cords. The swelling is sometimes unilateral, sometimes subglottic, and may be so extreme as entirely to close the glottis. The mucous membrane is swollen, pale, grayish, and semitranslucent, or dull reddish in color. Rarely the exudation is purulent.

Symptoms.—The symptoms are those of severe laryngitis, together with urgent dyspnœa—at first inspiratory and later expiratory stridor, inspiratory depression of the intercostal spaces, scorbiculus cordis, etc. Suffocation, that may lead almost immediately to a fatal issue, may be the first manifestation of the disease. The œdema is quite apparent on laryngoscopic examination; the swelling of the epiglottis may be detected by the finger or seen when the tongue is depressed.

Diagnosis.—The diagnosis is easy from the symptoms and the results of laryngoscopic examination or palpation by the finger.

Prognosis.—The prognosis depends upon the basic condition and the promptness with which treatment is instituted. The disease is often fatal.

Treatment.—The patient should be given ice to suck, an ice collar should be applied externally, the throat and larynx should be sprayed with cocaine and adrenalin chloride, and the swollen areas should be scarified. If the symptoms are urgent, tracheotomy should be performed immediately, since the high mortality is due to delay.

SPASMODIC LARYNGITIS.

(*Laryngismus Stridulus; Stridulous Laryngitis; Spasmodic Croup; False Croup.*)

Etiology.—Spasm of the larynx occurs in association with divers diseases of the larynx, but it is a dominant feature of the disorder known as spasmodic laryngitis or laryngismus stridulus. Probably these are distinct disorders, the one an acute catarrhal laryngitis with spasm, the other a pure neurosis. The disorder is peculiar to children, especially rickety children, between the ages of six months and six years, and is said to be more common in boys. It is closely related to tetany, and may be one of its manifestations. In some cases it is related to enlargement of the thymus and receives the name thymic asthma (page 403). The attacks are often provoked by passion or emotional disturbances—so-called "passion fits" and "attacks of holding the breath." Whether the exciting cause acts centrally, as is supposed in rickets, or peripherally, has not been determined. Among the causes believed to act reflexly are adenoids, teething, indigestion, preputial irritation, intestinal parasites, etc.

Pathology.—The lesions consist of paroxysmal tonic spasms of the adductor muscles of the glottis, in consequence of which the glottis is closed and breathing rendered impossible. There may or may not be an associated catarrhal laryngitis.

Symptoms.—The attacks usually come on at night, the child having gone to bed well and being awakened out of a sound sleep. As a rule, there are no premonitory symptoms, no cough, no hoarseness. The attack begins with a forced inspiration, followed by complete cessation of respiration. The face, at first pale, becomes cyanotic and anxious, the eyes protrude and are rolled about, violent inspiratory efforts are made, the intercostal spaces and the abdomen become retracted, and profuse perspiration breaks out. The attack may last for from several seconds to several minutes, when suddenly with relaxation of the contracted adductors, a deep labored, noisy inspiration ("child-crowing") occurs, the cyanosis and anxious expression disappear, and the child becomes comfortable. In some cases there is loss of consciousness and tonic and clonic spasms of the trunk and extremities (carpopedal spasms). Rarely the attack leads immediately to death. Attacks vary much in severity, and they may occur singly or be frequently repeated—during the day as well as at night.

Diagnosis.—The diagnosis is quite apparent from the symptomatology and the etiological factors. Membranous, or diphtheritic, laryngitis (true croup) may be distinguished by the presence of fever, the gradual development of the dyspnœa, and presence of a false membrane in the fauces or in the larynx on laryngoscopic examination.

Prognosis.—The prognosis is good, since despite its alarming aspects, the disease is rarely fatal.

Treatment.—During the paroxysm cold water should be dashed in the child's face, or the child should be placed in a warm bath (100° to

105°) and cold water applied to its back and chest. Recourse may be had to a few whiffs of chloroform, or an emetic, such as ipecac. Recurrence of the attacks may be obviated by cold or counterirritation to the neck, and the internal use of bromides, chloral, belladonna, etc. All possible sources of peripheral irritation must be corrected. Since the children are anemic and rachitic, tonics containing iron, arsenic, phosphorus, strychnine, cod-liver oil, etc., and nutritious diet must be given for a long time.

CHRONIC LARYNGITIS.

(*Chronic Laryngeal Catarrh; Preacher's Sore Throat.*)

Etiology.—Chronic laryngitis results from repeated attacks of acute laryngitis; the long continued and ill use of the voice (public speakers, preachers, singers, etc.); the continued action of injurious agents upon the larynx, such as tobacco smoke, dust (stone cutters, etc.), alcohol (inebriates), etc.

Pathology.—The lesions, as a rule, consist of swelling, thickening, and pale grayish or bluish-red discoloration of the mucous membrane, especially of the epiglottis, the interarytenoid folds, and about the vocal cords. Rarely superficial erosions or ulcerations occur. Special types of the disease are described: Laryngitis hypertrophica, or verrucosa—verrucous projections or swellings the result of hyperplasia of the epithelium and the papillæ; laryngitis atrophica—pallor and thinness of the mucous membrane the consequence of atrophy; and laryngitis hypoglottica chronica hypertrophica, or chorditis vocalis inferior chronica—hyperplasia of the subchordal mucous membrane, leading to marked constriction of the laryngeal lumen.

Symptoms.—The symptoms consist of irritation in the throat, cough, hoarseness, and expectoration—varying in severity from time to time. The sense of irritation compels the patient continually to attempt to clear the throat; the cough is rough, harsh; the hoarseness varies in degree to complete aphonia; and the expectoration is usually scanty, occasionally rather abundant, mucous, mocopurulent, and rarely blood-streaked.

Diagnosis.—The diagnosis is usually quite obvious from the symptoms, but recourse should be had to laryngoscopic examination to ascertain the exact conditions, and to exclude tuberculosis, syphilis, tumors, etc.

Prognosis.—The prognosis as to complete recovery is poor, but marked amelioration of the symptoms is possible. Relapses are common, especially since the etiological factors are not readily controlled.

Treatment.—Of prime importance is removal of the etiological factor—overuse of the voice, alcohol, tobacco, as the case may be. Change of climate—a sea voyage, residence in a pine forest, etc.—is often of the greatest benefit. Disease of the nose and throat must be corrected. Cold compresses externally are often of service.

Approved internal remedies comprise the following, which may be applied on a swab or by means of an atomizer: Tannic acid (1 to 2 per cent.), zinc sulphate (2 to 5 per cent.), potassium chlorate (2 to 5 per cent.), silver nitrate (1 to 2 per cent.), alum (0.5 to 1 per cent.), etc. Insufflation of powders, bismuth, etc., is sometimes of service, if done carefully. The general health often demands the administration of tonics.

LARYNGEAL PERICHONDRITIS.

Etiology.—Laryngeal perichondritis may be rarely primary—when it is usually due to trauma; commonly it is secondary and occurs during the course of syphilis, tuberculosis, typhoid fever, diphtheria, scarlatina, erysipelas, etc.

Pathology.—The cricoid and the arytenoid cartilages are most frequently affected; less commonly the thyroid and the epiglottis. Rarely the lesions are metastatic. Commonly there is at first more or less superficial ulceration of the mucous membrane—whence the lesions spread to the perichondrium. Usually the process goes on to abscess formation which gradually separates the perichondrium from the underlying cartilage; soon the cartilage becomes necrotic and separates into small particles which with the other contents of the abscess are discharged—internally (perichondritis interna) or externally (perichondritis externa). The defect is usually remedied by cicatrization and consequent stenosis; but when the abscess is discharged externally a fistula not uncommonly results.

Symptomatology.—Occasionally, especially in the primary affection, there may be an acute invasion, with chill (or chilliness) and fever. The characteristic symptoms consist of severe spontaneous pain, tenderness on pressure over the larynx, hoarseness, cough, painful dysphagia (especially when the posterior portion of the cricoid cartilage is involved), and the development of laryngeal stenosis which may reach an extreme and dangerous degree. Laryngoscopic examination reveals redness and swelling of the mucous membrane, in some cases considerable œdema or a smooth localized swelling corresponding to the abscess. One or both vocal cords may become immobilized. In external perichondritis the swelling may be quite obvious and palpable; fluctuation may be obtainable; and the pus may gravitate to the mediastinum. Chronic laryngeal stenosis due to cicatricial contraction is a common event in the subjects that recover.

Diagnosis.—The difficulty of making a satisfactory laryngoscopic examination often makes the diagnosis obscure; expectoration of a bit of cartilage often makes it conclusive. Tuberculosis, syphilis (gummas), and tumors, and in the external form, softened lymph nodes, must be excluded.

Prognosis.—Death may occur from rapidly developing stenosis or the rupture of an abscess into the larynx. In subjects that recover,

stenosis of the larynx and some impairment of the voice are common.

Treatment.—In the acute stage, if stenosis is not marked, attempts may be made to reduce the swelling by the external use of an ice collar or leeches, and the internal use of hot drinks or hot inhalations, astringents, or scarification of the mucous membrane. Tracheotomy or laryngotomy is often necessary, although the abscess may sometimes be opened internally, with favorable results. The patient's general health must be maintained by careful feeding and stimulants. In tuberculous and syphilitic cases the general disease should be treated. In chronic stenosis dilatation may prove useful, or the patient may be obliged to wear a tracheal cannula.

TUMORS OF THE LARYNX.

Pathology.—Tumors of the larynx may be benign or malignant. The *benign tumors* occur at all ages, but are most common in adult life, and in males rather than in females. They comprise: papilloma (pinkish or grayish wartlike, sessile or pedunculated growths, often multiple); fibroma (spheroidal, smooth, firm, dense growth, usually single); mucous polyp, soft fibroma, myxofibroma (soft, translucent growth); chondroma (firm, hard, smooth); lipoma (rare, soft, yellowish); angioma (reddish, hemorrhagic); and cysts (small, transparent, fluctuating growths, usually retention cysts). Papilloma and fibroma are the most common, especially in children. Benign growths usually spring from the vocal cords, except the mucous polyp and cysts which are often found in the ventricles of Morgagni and on the epiglottis. The *malignant tumors* comprise sarcoma and carcinoma. Sarcoma is rare; occurs, as a rule, early in life; and forms soft or hard, smooth or diffuse infiltrations, sometimes verrucose growths, that commonly spring from the vocal cords. Carcinoma is more common; occurs, as a rule, in middle life or later; and forms wart-like excrescences, diffuse infiltrations, or soft, nodular, rapidly ulcerating growths, that involve the vocal cords, the ventricles of Morgagni, or the epiglottis. Sometimes the carcinoma is secondary to carcinoma of the tongue, pharynx, or œsophagus.

Symptomatology.—Of *benign tumors,* the most common symptoms are alterations in the voice (harsh, croaking, aphonia); cough (sometimes croupy in children); dyspnœa, varying in grade, depending upon the size and situation of the growth, and sometimes paroxysmal when the growth is pedunculated and mobile; orthopnœa, sometimes progressing to asphyxiation, from sudden occlusion of the glottis; and dysphagia, when the upper surface of the epiglottis is involved. Pain is often inconspicuous.

Of *malignant tumors,* the symptoms are quite like the foregoing. In some cases the voice is not affected—when the posterior surface of

the cricoid cartilage, the epiglottis, or the aryteno-epiglottidean fold alone is involved. Hoarseness, when it does develop, may be due to the growth itself, or to immobility of the vocal cords. When the epiglottis or the posterior surface of the larynx is involved, dysphagia is usually marked, and there may be increased salivation, a fetid secretion (sometimes blood-streaked, from ulceration), and an offensive breath. Pain radiating to the ear or the temporal region may be present. The cervical lymph nodes may become enlarged from metastases. Cachexia develops when the growth has become extensive, or when it is primary elsewhere.

Diagnosis.—Tumors of the larynx are to be diagnosticated only by laryngoscopic examination. Decision as to malignancy frequently depends upon removal of a portion of the growth and its study histologically. Benign tumors, as a rule, are pedunculated, multiple, do not infiltrate, show little tendency to bleed or ulcerate, and do not recur after removal. Rapid growth and recurrence after removal are significant of malignancy. Malignant tumors may be distinguished from syphilis and tuberculosis by the age of the patient, definite tumor formation, its primary localization to one side, the early immobility of the vocal cords, early ulceration, tendency to bleeding, pain, glandular involvement, the absence of tubercle bacilli in the secretions, and the nonresponse to antisyphilitic treatment.

Prognosis.—The prognosis in benign tumors is good, both with reference to life and the preservation of the voice. In malignant tumors the prognosis, as a rule, is bad, although in some cases the growth may be removed by early operative procedures.

Treatment.—The treatment of tumors of the larynx is surgical. Most benign tumors may be removed by intralaryngeal methods under local anesthesia. In children this is difficult, and thyrotomy, subhyoid pharyngotomy, or laryngotomy may be necessary. In malignant tumors partial or complete laryngectomy offers the only hope of success. When metastasis has occurred laryngectomy is usually useless; the patient may be made comfortable by tracheotomy and the subsequent use of a tracheal cannula.

DISEASES OF THE TRACHEA AND THE BRONCHI.

ACUTE BRONCHITIS.

(*Acute Bronchial Catarrh; Acute Tracheitis; Acute Tracheobronchitis.*)

Etiology.—Acute bronchitis may occur at any age, but it is probably most common during adolescence and adult life, although it is more serious and fatal in the very young and the very old. It is more common in males than in females, on account of their mode

of life and occupations. It prevails especially during changeable (moist and cold) weather—the late fall, the winter, and the early spring. Sedentary life, working in close, ill-ventilated rooms, debility from any cause predispose to the disease; an out-of-door life is a preventive. It may occur as a primary, independent affection, or in association with (or secondary to) other disorders. The exciting causes of the primary disease are the infectious, toxic, mechanical, and thermic irritants provocative of acute catarrhal laryngitis (page 517); indeed, the one often follows the other, and both result from "catching cold." Divers bacteria appear to be the most common cause. As a secondary disease, it occurs in many infections, such as measles, scarlatina, pertussis, diphtheria, influenza, typhoid fever, malaria, syphilis, tuberculosis, etc.; it is common in the subjects of chronic heart disease, chronic lung disease, Bright's disease, gout, etc. Some persons seem unusually susceptible (hereditary influences?).

Pathology.—The mucous membrane of the trachea and bronchi is red, swollen, and covered with mucus and mucopus; occasionally there may be punctiform hemorrhages. The lesions may extend to the finer bronchi—and collapse, bronchopneumonia, emphysema, or œdema and congestion of the lungs may result. The microscopic lesions consist of inflammatory hyperemia of the mucosa, œdema and cellular infiltration of the submucosa, and swelling and desquamation of the lining epithelial cells.

Symptomatology.—Depending upon the extent and intensity of the manifestations, three forms of acute bronchitis may be distinguished: (1) A mild form, in which the lesions are confined to the trachea and the main bronchi; (2) a more severe and more diffuse form, in which the medium-sized and the smaller bronchi also are involved; and (3) a form in which the finest bronchi and bronchioles are involved—capillary bronchitis (identical with bronchopneumonia). In rare cases, the onset is abrupt, and attended by a chill; chilliness is not uncommon. In many cases an acute coryza precedes the bronchitic phenomena; laryngeal symptoms also may be associated.

In the mild form the symptoms consist of substernal distress, pain, or a sense of rawness or oppression, and cough, at first hard, harsh, often paroxysmal and painful, especially beneath the sternum and along the attachment of the diaphragm. In the beginning there may be no expectoration, or with considerable effort a small amount of tough, viscid, occasionally blood-streaked mucus may be expelled; after a few days, however, the sputum becomes more abundant, less viscid, and mucous or mucopurulent, in character. There may be little or no general disturbance, though commonly there is some languor and headache, possibly, but not always, some elevation of the temperature. In the more severe forms, the general and the local symptoms are aggravated. Languor, weariness, and headache are usually more marked; general neuromuscular pains are often quite severe; and the temperature may be 102° F., or thereabouts, for at least a day or two.

The cough may be extremely distressing, but, as in the milder form, it becomes much ameliorated with the development of free expectoration. There may be some dyspnœa and acceleration of the pulse rate. Cyanosis, not uncommon in the young and the aged, suggests capillary bronchitis (bronchopneumonia).

Physical Signs.—In the mild form, when the lesions are confined to the trachea and the main bronchi, there may be no abnormal physical signs, aside from harshness of the breath sounds and some prolongation of the expiration. In the more severe form, in addition to the foregoing, sonorous and sibilant rales may be heard, especially at the bases posteriorly, in the early stages, and fine and coarse bubbling rales later when the bronchial secretion has become freer. In children there are often evidences of interference with free ventilation of the lungs—dyspnœa, inspiratory retraction of the lower end of the sternum and the lower ribs, use of the accessory muscles of respiration, etc.

Diagnosis.—The diagnosis, as a rule, is easy. The bilateral distribution of the physical signs is important. One should not overlook the fact that bronchitis may be the initial manifestation of certain infectious diseases, such as measles, pertussis, typhoid fever, malaria, etc. A history of exposure to any of these infections is valuable. The sputum should be examined to determine the nature of the infecting agent. Persistence of fever for more than several days suggests something more than mere bronchitis. Capillary bronchitis (bronchopneumonia) is indicated by severe general symptoms, fever, dyspnœa, and cyanosis. Croupous pneumonia should be readily differentiated by the absence of the characteristic physical signs. Predominance of fine rales at the apices, rather than at the bases, suggests tuberculosis—even in chronic bronchitic subjects. The diagnosis of the secondary form of bronchitis presupposes the recognition of the primary cardiac, pulmonary, renal disease, etc.

Prognosis.—Except at the extremes of life, a fatal termination is rare. Mild cases usually subside within a week, and convalescence is established at the end of two weeks. In severe cases the course is more protracted. One attack often predisposes to another. In the very young and the aged, in emphysematous, cardiac, and nephritic subjects, the disease is commonly serious and may lead to a fatal termination.

Treatment.—Although not always possible, it is desirable that the patient go to bed for a day or two; at least, he should remain at rest in a well-ventilated room, and his diet should be restricted. The attack may sometimes be aborted by Dover's powder (10 grains, 0.6 gram), hot lemonade (with or without whiskey), and a hot mustard footbath at bed time, followed in the morning by calomel or blue mass and a saline laxative in adults, or castor oil in children. A mustard plaster, or turpentine stupe applied to the chest serves to relieve the local tightness and soreness; or the chest may be rubbed with camphorated oil or camphor liniment.

In the early stages much relief is afforded by a sedative diaphoretic mixture, such as the following:

℞—Spirit of nitrous ether	3 drams	12|00
Wine of ipecac	1½ drams	6|00
Potassium citrate	3 drams	12|00
Cinnamon water, sufficient to make	3 ounces	100|00—M.

S.—Two teaspoonfuls (10 c.c.) every three hours (to an adult).

If the pulse is very rapid or there is much fever, minim doses of tincture of aconite may be added to each dose (especially serviceable in children). Additional relief to the dryness of the throat and the difficulty in breathing is afforded by inhalations of steam impregnated with sodium chloride, sodium bicarbonate, compound tincture of benzoin, etc.; or the Dover's powder may be repeated at night, or codeine or heroin substituted, but discretion must be exercised in the use of these remedies in the young and the aged.

If, after several days, the cough continues troublesome and the expectoration has not become free, the following old-fashioned mixture will be found efficacious:

℞—Ammonium chloride	1½ drams	6|00
Syrup of squill	3 drams	12|00
Compound mixture of glycyrrhiza	3 ounces	100|00—M.

S.—Two teaspoonfuls (10 c.c.) four times daily.

Paroxysms of coughing (referable to bronchial spasm) may be controlled by belladonna, stramonium, or lobelia; belladonna is efficacious also if the expectoration is copious and continues for a long time—in which event the balsamic preparations also are useful—such as terebene, terpene hydrate, tar, etc. In children, if the expectoration is abundant and not well raised (provoking dyspnœa and cyanosis) an emetic should be administered—alum (20 grains, 1.3 gram) or wine of ipecac (1 dram, 4 c.c, repeated in ten minutes, if necessary). Signs of prostration and a weak heart call for the administration of stimulants. During convalescence, nutritious diet, fresh air, and tonics, stomachics, quinine, strychnine, iron, and arsenic, should be given.

CHRONIC BRONCHITIS.

(Chronic Bronchial Catarrh; Chronic Catarrhal Bronchitis; Winter Cough.)

Etiology.—Chronic bronchitis is rare as a primary disorder; but it may occur as a result of repeated acute attacks, or it may be chronic from the outset in consequence of the continued inhalation of irritating dust, vapors, etc. It is not uncommon in the middle aged and the aged, recurring each year with the cold, damp, and changeable fall and winter weather. Often it is secondary to chronic cardiac, pulmonary, or renal disease. It is also a manifestation of gout.

Pathology.—The condition of the bronchial mucous membrane varies in different cases. Often it is thickened irregularly, hyperemic, and covered with mucopus; in places the epithelium may be entirely wanting—granular patches. The fibrous and muscular coats are usually thickened and the seat of cellular exudation. In long-continued cases more or less atrophy occurs, and leads to cylindrical dilatation (bronchiectasis) or diverticula. Emphysema, fibroid induration of the lung, and hypertrophy of the heart (especially of the right ventricle) are common concomitants.

Symptomatology.—The principal symptoms are cough, expectoration, and dyspnœa—which vary much in different cases, and permit of the differentiation of several types of the disease.

Chronic Winter Cough.—This is probably the most common variety, and is characterized by the recurrence each winter of cough, which usually is not very troublesome, and expectoration, moderate in amount, mucous or mucopurulent in character. These usually lessen or disappear during the late spring, the summer, and the early autumn.

Chronic Bronchitis with Emphysema.—This is very common, and usually results from repeated attacks of acute bronchitis or of recurrent attacks of winter cough. The cough is often troublesome; it is more marked in the morning (accumulation of secretion during the night), and may be violent or paroxysmal; it commonly lessens or disappears during the summer. The expectoration may be scanty, viscid, and difficult to raise; or it may be abundant, and mucopurulent or seropurulent in character; occasionally nummular. Substernal distress, and more or less dyspnœa (especially after coughing or exertion) are common. The general health is often maintained for years.

Dry Chronic Catarrh (Catarrh Sec) of Laënnec.—This is characterized by a dry, troublesome, paroxysmal cough, dyspnœa, tightness of the chest and very scanty expectoration, which often contains grayish, pearl-like particles that are raised with difficulty. Emphysema is often associated; asthmatic attacks occur from time to time; and the subjects are prone to attacks of acute bronchitis.

Gouty bronchitis is an obstinate form, sometimes associated with other manifestations of gout. Dyspnœa is often extreme. Marked pulmonary congestion may lead to the expectoration of blood-stained sputum. There is usually associated disease of the heart, liver, and kidneys.

Fetid (or *putrid*) *bronchitis,* although usually seen in association with gangrene or abscess of the lung, bronchiectasis, the decomposition of the contents of tuberculous cavities, the rupture of an empyema or subdiaphragmatic abscess into the lung, etc., sometimes occurs as an independent affection. The cough is usually paroxysmal and severe; the sputum is usually abundant (upward of a pint in twenty-four hours), malodorous, thin, seropurulent in character, and on standing separates into three layers—the lowermost consisting of purulent material, detritus, and irregular masses the size of peas (Dittrich's plugs); a middle layer

of discolored fluid; and an upper layer of frothy mucus. Fever, loss of appetite, and other manifestations of bacterial intoxication are not uncommon. Should the disorder be primary, secondary changes, such as gangrene or abscess of the lung, septic metastases (as in the brain, etc.), may occur.

Bronchorrhœa may follow long continued bronchitis of the ordinary type. The cough and expectoration often occur in paroxysms, and are associated with extreme dyspnœa. The expectoration is very abundant—as much as one or two quarts in the twenty-four hours; it is usually thin, frothy, serous or mucoserous, occasionally somewhat purulent or hemorrhagic, rarely thick and distinctly purulent, in character. The condition may lead to permanent dilatation of the bronchi, and fetid bronchitis. In the young especially it often persists for a long time without noteworthy impairment of the general health.

Physical Signs.—The physical signs commonly include those of emphysema. In the absence of emphysema the percussion note is resonant, as a rule; there may be some impairment, especially posteriorly, in the event of there being much secretion, collapse of small areas of the lung, thickening of the pleura, etc. The breath sounds are usually harsh and vesicular in character, the expiration prolonged; and there are rales, sibilant and sonorous, or fine and coarse bubbling, or crackling, especially posteriorly, depending upon the conditions within the bronchi. In the dry catarrh no rales are present. Evidences of hypertrophy and dilatation of the right ventricle may also be detected.

Diagnosis.—As a rule, the diagnosis is easy. Care must be exercised to exclude pulmonary tuberculosis (repeated examination of the sputum, etc.), and one must remember that a chronic bronchitic subject may become tuberculous. Distinction must be drawn between the primary cases and those due to chronic cardiac, pulmonary, and renal conditions. Aortic aneurysm, mediastinal and pulmonary tumors, etc., have been mistaken; but the physical signs, if sought for, are usually conclusive.

Prognosis.—Chronic bronchitis, as a rule, is a very obstinate disease, tends to recur, and to become worse with advancing years; but recovery is not impossible, and marked amelioration of the symptoms is readily attained. The prognosis is good in young subjects, and in older subjects able to live in a suitable climate. The prognosis is bad in subjects of chronic heart, pulmonary, and renal disease, and in the aged.

Treatment.—Removal to a warm, moist, equable climate (Florida, Southern California, Riviera, etc.) constitutes the most successful treatment—prophylactic as well as curative. Those unable to avail themselves of this should keep their room warm, though well ventilated, live outdoors as much as possible in sunny, pleasant weather, avoid vitiated atmospheres; in a word, avoid the factors known to provoke a "cold." The diet should be nutritious and easily assimilable. In patients the subject of chronic cardiac, pulmonary, or renal disease, gout, etc., the primary disorder must receive careful and appropriate treat-

ment. Thus, some patients are much improved by small doses of digitalis, strophanthus, convallaria, and other heart tonics; attention to free elimination by the kidneys and free evacuation by the bowels materially improve others; whereas, the obese and the gouty often respond markedly to a proper dietetic regimen. Attention to the general health, and the use of cod-liver oil, arsenic, iron, quinine, and strychnine are followed by good results in other cases.

Medicinal treatment directed to the bronchitic condition must be varied to suit the exigencies of each individual case. In some cases the treatment need not be different from that appropriate in the later stages of acute bronchitis. Often potassium (or ammonium or sodium) iodide is of marked benefit, especially when the cough is paroxysmal and associated with emphysema; often it may serviceably be combined with ammonium chloride (or carbonate) and lobelia; or if secretion is free, with squill or senega; but the use of the iodide should not be too long continued. In most cases the balsamic preparations are of much value—oil of sandalwood, of eucalyptus, of turpentine, and of copaiba; balsam of Peru or of tolu; terpene hydrate, terebene, tar, creosote, etc. I have seen much benefit from 30 minims (2 c.c.) each of syrup of tar and syrup of wild cherry, four times a day. If the cough is distressing at night, resort must often be had to opium or its derivatives, codein, heroin. The troublesome morning cough may sometimes be relieved by the use of alkalies (sodium chloride, sodium bicarbonate, potassium acetate, etc.) in hot water. The dry distressing cough may be benefited by inhalations of steam impregnated with sodium chloride, tincture of benzoin, oil of eucalyptus, white pine, etc. In fetid bronchitis the odor may be improved by sprays of carbolic acid (0.05 to 0.1 per cent.), thymol (0.2 to 0.5 per cent.), or by resort to intratracheal medication. Severe dyspnœa and pain in the chest may be relieved by local applications to the chest, such as plasters, dry or wet cups, hot or cold compresses, etc., or by venesection. Methodic compression of the thorax during expiration, the use of the pneumatic cabinet, and posture that favors the exit of secretion are of service in some cases.

FIBRINOUS BRONCHITIS.

Etiology.—Fibrinous bronchitis, a disease attended by the formation of so-called fibrinous casts of the bronchi, may be primary or secondary. The primary is an independent disorder, rare, and of obscure etiology. It occurs about middle life, twice as frequently in men as in women, prevails during the winter and spring, and tends to recur from year to year. It may affect persons previously well or the subjects of bronchitis or tuberculosis. The secondary form has been observed in certain infectious diseases, such as diphtheria, scarlatina, measles, pneumonia, influenza, typhoid fever, etc.; in certain chronic cardiac and pulmonary diseases; in diseases of the skin, such as pemphigus, herpes, impetigo,

etc.; complicating menstruation and pregnancy; etc. Casts of the bronchi have also been seen in association with the albuminous expectoration that sometimes follows tapping a pleural effusion; and blood casts occur with hemoptysis. These secondary forms are not at present under consideration.

Pathology.—The characteristic lesion consists of the formation of membranous, partly hollow, partly solid, casts of the bronchi, which, dividing dichotomously, may reach to the finest bronchi. The subjacent mucous membrane may appear intact, or its epithelium may be shed and it may show the usual evidences of chronic bronchitis. The lesions are often curiously confined to definite areas, which in the recurring form are sometimes repeatedly and only involved. The casts are grayish white in color, occasionally covered with streaks of blood, and vary much in size from small irregular pieces to masses measuring six to eight inches in length. On transverse section they present a concentric appearance. Usually said to be fibrinous, recent investigations seem to show that they are made up mostly of mucin; some observers believe them to consist of syntonin or coagulated albumin. Microscopically the ground substance contains leukocytes (mostly mononuclear), epithelium, sometimes erythrocytes, Charcot-Leyden crystals, and Curschmann's spirals. Various microörganisms have been cultivated from the membrane, especially pneumococci, streptococci, and staphylococci. The nature of the process is ill understood, but it is sometimes considered analogous to mucous colitis. The rare disorder known as acute obliterating fibrinous bronchiolitis is doubtless of like nature.

Symptomatology.—Acute and chronic forms of the disease may be distinguished. The acute form is rare, and usually begins suddenly with a chill (or chilliness), fever, cough, expectoration, and dyspnœa. At first and for several days, as a rule, the attack resembles an ordinary bronchitis; soon, however, the cough becomes severe and paroxysmal; the expectoration, which heretofore has been mucous in character, is raised with considerable difficulty; dyspnœa becomes urgent, and eventually with a more or less violent effort the characteristic cast is expelled—with great relief to the patient. The expectoration again becomes more abundant and may contain small coagula. There is sometimes associated hemoptysis; rarely a large hemorrhage. The attack may last several days or several weeks.

The chronic form may develop out of a chronic bronchitis or tuberculosis, and between attacks there are usually the ordinary evidences of bronchitis. The attacks are less severe than in the acute form, and they recur at longer intervals, often yearly; there is usually little or no fever, and the dyspnœa is less urgent. Should the attacks recur at short intervals, small coagula are likely to be expectorated frequently and the general health of the patient may deteriorate.

Physical Signs.—The physical signs are those of bronchitis. Over the portion of lung affected there may be weakened or absent breath

sounds, and fine bubbling rales may be provoked by voluntary coughing. After the loosening of the cast, before its expectoration, a rough clicking sound (bruit de drapeau, Ventilgerausch) is said to be audible.

Diagnosis.—The diagnosis depends upon the detection and recognition of the casts. These may be small and escape observation. Their detection is facilitated by floating them on water. Severe paroxysms of coughing and dyspnœa relieved by expectoration are suggestive, and should lead one to search for the casts. Care must be taken to exclude the secondary form of the disease.

Prognosis.—The prognosis in acute cases is grave, death occurring in from one-third to one-half the cases from suffocation or bronchopneumonia. The chronic variety may last for many years, but eventually recovery may ensue. Tuberculosis, emphysema, atelectasis, pleuritis, and chronic heart disease are rather common sequels.

Treatment.—In the acute cases, the treatment is that of acute bronchitis; and the only hope of cure in the chronic cases is the cure of the commonly associated chronic bronchitis. Inhalations of atomized sodium carbonate or bicarbonate (2 to 5 per cent.), lime water, or compound tincture of benzoin, mitigate the distress and are said to assist in loosening the membrane. Pilocarpin, since it promotes free bronchial secretion, is useful for the same purpose. Potassium iodide is probably of even more value. Mercurial inunctions also have been recommended. In the event of marked dyspnœa, cyanosis, and delayed expectoration, emetics should be resorted to.

BRONCHIECTASIS.

Etiology.—Bronchiectasis, dilatation of the bronchi, may be due to a congenital defect—in which event it is likely to be universal; otherwise, it results from weakening of the bronchial wall, which, in consequence, becomes unable properly to withstand the distending force of the contained air (much exaggerated during paroxysms of coughing). Acland has well classified the causes, as follows: (1) In the lumen of the bronchial tubes, (*a*) the impaction of a foreign body, and (*b*) the inhalation of fibrinous plugs, as in diphtheria; (2) (*a*) acute affections of the walls of the bronchial tubes, resulting from pneumonia, bronchopneumonia, tuberculosis, diphtheria, and whooping cough, and (*b*) chronic affections of the wall of the bronchial tubes, such as chronic bronchitis (60 per cent. of the cases), syphilitic peribronchial fibrosis with stenosis, and tuberculosis; (3) affections of the parenchyma of the lung, such as chronic interstitial pneumonia (10 per cent. of the cases), and collapse from causes other than pressure; (4) affections external to the lung, such as aneurysm, neoplasm, and pleural adhesions and thickening (12.5 per cent. of the cases). The disease is more common in males than in females. About 50 per cent. of the cases occur between the twentieth and the fortieth year; 23 per

cent. between the tenth and the twentieth year; and 13 per cent. between the fortieth and the fiftieth year.

Pathology.—The bronchiectatic cavities may be cylindrical, fusiform, saccular, or moniliform, but the cylindrical and the saccular are the more common, and may exist together. The condition may be general—in which event it is unilateral, rarely congenital, and commonly follows pneumonia; or it may be partial—in which event it is bilateral and occurs in tuberculosis (especially at the apex), pleurisy (especially at the base), and emphysema. In the general form the entire bronchial tree consists of a series of sacculi communicating the one with the other, lined with a smooth, glistening mucous membrane (frequently transition of cylindrical epithelium into flat), except in the dependent portions where there may be some ulceration; the dilatations are sometimes extreme, forming large cysts that may extend to the pleural surface; the intervening pulmonary tissue is indurated and fibrotic. In the partial form, the mucous membrane is inflamed and ulcerated and the muscular tissue thin and atrophic; cicatrization may give rise to stenosis of the lumen. The contents of the cavities are often horribly fetid, and on culture reveal divers pyogenic microörganisms, the influenza bacillus, etc.

Symptomatology.—Most of the symptoms are those of the chronic bronchitis, emphysema, tuberculosis, etc., with which the bronchiectasis is associated; in many of these cases the existence of the bronchiectasis is not even suspected. The characteristic symptoms consist of: (1) Cough, which, from time to time, especially in the morning or the evening, or on sudden change of posture, becomes exaggerated into violent paroxysms, and results in the expulsion of (2) usually a large amount (500 c.c. or more) of grayish, acid, fetid, mucopurulent sputum, which on standing separates into three layers—the lowermost, dense, purulent, and granular; the middle, watery and sometimes discolored; and the uppermost, frothy, discolored, and mucous. Microscopically, it consists of mucus, pus, detritus, fatty acid and hematoidin crystals, elastic fibers (when ulceration has occurred), bacteria, etc. Sometimes it contains a notable amount of blood; sometimes large hemorrhages occur. (3) Fetor of the breath, especially on coughing (it may be missed if the cavities are in the apex and well drained). (4) The physical signs of cavity formation. The temperature is variable; it may be normal even when the sputum is fetid; but in the event of necrosis of the bronchial wall and toxic absorption, it corresponds to the septic type. Dyspnœa and cyanosis on exertion are common.

An acute and fatal form of the disease is not uncommon in children (bronchiolectasis), and is usually associated with bronchopneumonia.

Complications and Sequels.—Complications and sequels are many. Pneumonia, abscess and gangrene of the lung, interstitial pneumonitis, empyema, pyopneumothorax, etc., are common; septicemia, pyemia, metastatic abscesses (brain, liver, kidney), clubbing of the finger tips,

hypertrophic pulmonary osteo-arthropathy, amyloidosis, etc., are not uncommon.

Diagnosis.—In the absence of the characteristic symptoms the disease often escapes clinical recognition. Tuberculous cavities, as a rule, are more common at the apex, preceded by signs of solidification, progressive in character, and the expectoration contains tubercle bacilli; whereas, bronchiectatic cavities are more common at the base, followed by induration of the lung, often non-progressive, and the expectoration does not contain tubercle bacilli; but tuberculosis may supervene upon non-tuberculous bronchiectasis. Fetid sputum with normal temperature is almost certainly from a bronchiectatic cavity.

Prognosis.—Cure is extremely unlikely, if it occurs at all; but the patient may live for many years and lead a fairly active life. Death usually results from septicemia, emaciation, or one of the complications or sequels. The acute form is usually fatal from asphyxia.

Treatment.—Attention to the primary disorder and the general health of the patient are of prime importance. The best medicinal treatment consists of the inhalation of vaporized creosote, for fifteen minutes every other day at first, later every day. A dram or two of crude creosote should be poured on some water contained in a saucer and slowly vaporized over an alcohol lamp. This often at first provokes coughing, which has the effect of ridding the bronchial tubes of retained secretion and then permitting the local action of the creosote on the mucous membrane. Inhalations of carbolic acid, eucalyptus, menthol, etc., are of much less value. Intratracheal injections are sometimes of service; Grainger Stewart's is one of the best: Menthol, 10, guaiacol, 2, and olive oil, 88, of which two to three drams (8 to 12 c. c.) should be injected at a time. There is much more likelihood of good effect if the injection follows soon upon a copious expectoration—cleansing of the bronchi. The internal use of balsamic preparations, oil of sandalwood, terebene, terpene hydrate, etc., should be tried. The adoption of postures that favor drainage of the cavities is of value. Surgical drainage has been practised in some cases with good results.

BRONCHIAL ASTHMA.

(*Spasmodic Asthma.*)

Bronchial asthma is a disorder characterized by attacks of dyspnœa (chiefly expiratory) and a characteristic expectoration; it commonly occurs in bronchitic subjects, and ultimately leads to emphysema.

Etiology.—Asthma is not infrequently familial; it is twice as common in men as in women; it may occur at any age; and it sometimes develops during childhood and persists throughout life. The attacks are excited by divers stimuli—emotion, fright, unpleasant odors, irritating dust, certain articles of diet, too much food (overloaded stomach), cold and damp atmosphere, etc. Some subjects are freer while in the country,

others while in the city. The disorder has much in common with hay-fever, and both may occur at the one time or alternate in the same subject.

Pathology.—The nature of the disorder is not well understood. In many cases it seems to be chiefly, if not wholly, a neurosis, and due to spasm of the bronchial muscles or swelling of the bronchial mucous membrane, in consequence of disorder innervation—pneumogastric or vasomotor. Others attribute the phenomena to spasm of the diaphragm, of all the inspiratory muscles, or of the musculature of the pulmonary arteries. In many cases, however, and this is especially important, although frequently overlooked, there is inflammation of the bronchioles (bronchiolitis exudativa of Curschmann), attended by swelling of the mucosa, the exudation of a viscid mucus, and desquamation of the surface epithelium—which assuredly suffice to explain the phenomena. In some cases there is associated disease of the nose (hypertrophic rhinitis, polyps, adenoids), of the larynx and trachea (catarrh, enlarged bronchial lymph nodes), disease of the genitalia, etc., which often apparently stand in causal relationship to the asthma. Most subjects reveal the lesions of chronic bronchitis; others are gouty, nephritic, etc.

Symptomatology.—The essential symptoms consist of attacks of dyspnœa followed by the expectoration of a characteristic sputum. Though usually not, the attacks are sometimes preceded by premonitory symptoms (a sense of constriction or oppression of the chest, flatulence, etc.), aura asthmatica. They commonly come on during the night, the patient being awakened from a sound sleep by dyspnœa and a sense of suffocation that soon become extreme and often induce him to seek an open window in the hope of relief. The chest soon becomes fixed in full expansion, the accessory muscles of respiration become tense and make violent, though useless, efforts, and the expiration becomes much prolonged, labored, and accompanied by loud wheezing. The respirations, as a rule, are not much increased in frequency; the pulse is small and accelerated, the temperature is usually normal or subnormal, the extremities become cool, the face livid, the expression anxious, and sweat not infrequently breaks out.

Early in the attack there is usually little or no cough or expectoration. After several hours, however, sputum begins to be expectorated; this consists of a small amount of viscid mucus containing roundish gelatinous masses (perles of Laënnec) which represent casts of the bronchioles (bronchiolitis); many of these have a twisted spiral arrangement (Curschmann's spirals), and may or may not reveal a central translucent filament. In addition, the sputum contains leukocytes (mostly eosinophilic), epithelium from the bronchi and the alveoli, and Charcot-Leyden octahedral crystals. Examination of the blood usually reveals an eosinophilia (25 to 50 per cent.).

A single attack may last from upward of a half hour to several hours, or the patient for days, sometimes for several weeks, may partially

recover and relapse, especially at night. At the end of several days in severe attacks, sooner in milder attacks, the sputum becomes more abundant, mucopurulent in character, and Curschmann's spirals disappear. At this time the patient's general distress becomes much lessened, although he is likely to remain weakened, apathetic, and dyspnœic for some time; indeed, more or less dyspnœa is not uncommon. The patient may remain free from attacks of asthma for weeks, months, or years at a time.

Physical Signs.—During the asthmatic attack the chest is usually in full inspiratory expansion and fixed; inspiration is short and jerky, the movements of the chest are altogether disproportionate to the efforts of the accessory muscles of respiration; the diaphragm is depressed and almost immobile; the percussion note is normal or hyperresonant (especially in long-standing cases in which emphysema develops); on auscultation the expiration is prolonged, and there are sibilant and sonorous rales. Later the rales become crackling or fine or coarse bubbling.

Diagnosis.—The diagnosis is usually easy; the symptoms, the characteristic sputum, and the physical signs distinguish asthma from other disorders attended by dyspnœa. Uremic asthma is differentiated by the signs of nephritis, high tension pulse, albuminuria, hypertrophy of the left ventricle (hypertrophy of the right ventricle occurs in long-standing asthma). Cardiac asthma, hysterical asthma, hay fever, spasm of the glottis, etc., sometimes must be considered the differential diagnosis.

Prognosis.—The prognosis is fairly good in early life, especially if a local cause (adenoids, nasal polyps, etc.) can be found and remedied. Recovery can scarcely be expected in middle and advanced life when chronic bronchitis, emphysema, hypertrophy and dilatation of the heart, etc., have developed, but the patient's suffering may be much ameliorated and he may live to a ripe old age. Although the individual attacks appear to be extremely severe, death during an attack is very rare.

Treatment.—Careful attention must be given to all the circumstances of the patient's life. Some do much better in the city, despite its smoke, and dirt, and soot, than in the purer atmosphere of the country; but climatic treatment must be instituted with reference to the response of each individual patient. The diet must be carefully regulated: the gouty and the obese must be abstemious; and in all cases, particularly in those disposed to flatulency, it is wise to advise the heavy meal in the middle of the day, and the partaking of very little food toward evening. The bowels should be opened at least once daily. Sleeping in a room the atmosphere of which is laden with steam (impregnated or not with the vapor of oil of eucalyptus, oil of pine, etc.) or with the fumes of burning nitre may secure freedom from nocturnal attacks and a consequent peaceful sleep. All sources of local irritation (nasal, pharyngeal, etc.) must be remedied.

During the attack relief may be obtained by the inhalation of amyl nitrite or chloroform, or the fumes evolved by burning the leaves (cigarettes) of belladonna, stramonium, lobelia, hyoscyamus, tobacco, or by burning paper impregnated with potassium nitrate; by drinking hot coffee and whiskey or spirit of chloroform; or by the hypodermic use of morphine (combined with atropine or cocaine), or pilocarpine—to be employed with caution. Tincture of lobelia in dose sufficient to produce the physiological effect is often efficacious, but it should not be employed when the heart is weak. An emetic sometimes assists in expelling retained secretions. An imminent attack may sometimes be warded off, or aborted, by the prompt use of amyl nitrite, nitroglycerin ($\frac{1}{50}$ grain, 0.0013 gram, hypodermicly), or chloroform.

Between the attacks no drug is so useful as potassium (or sodium) iodide; it has rendered me the best service when combined with lobelia. From time to time one of the bromides also may be added. Nitroglycerin is quite useful in some cases, especially in those with high arterial tension. Adrenalin chloride (hypodermicly), euphorbia pilulifera, arsenic, the compressed air of the pneumatic cabinet, oxygen, etc., appear to have been useful in some cases. When the heart is weak strophanthus or digitalis, in small doses, is serviceable.

TRACHEAL AND BRONCHIAL OBSTRUCTION.

Etiology.—Obstruction of the trachea or the bronchi may be due to: (1) A foreign body in the lumen; (2) stenosis the result of disease of the wall; and (3) compression from without.

Foreign bodies, depending upon their size, may cause partial or complete obstruction. Usually the obstruction is incomplete and bronchial rather than tracheal. The foreign body commonly passes through the trachea and is arrested in a bronchus, the right usually, since it is larger and more in a direct line with the trachea than the left.

Stenosis due to disease of the tracheal or bronchial wall may be caused by syphilis (usually), tuberculosis, leprosy, polyps, carcinoma, sarcoma, ecchondroma, etc. When due to syphilis, the stenosis may be annular, but it is also often somewhat longitudinal. The new growths may be situated anywhere, but they are more common in the bronchi near the bifurcation of the trachea.

Compression of the trachea or bronchi from without may be caused by tumors and other enlargements of the thyroid or thymus; enlargements of the regional lymph nodes (tuberculosis, leukemia, Hodgkin's disease, sarcoma, carcinoma), aneurysm, mediastinal abscess, Pott's disease or tumors of the vertebræ, etc.

Symptomatology.—The symptoms vary with the site and degree of the obstruction. A foreign body of sufficient size may lodge suddenly in the trachea, and cause complete obstruction and immediate death from asphyxia. A small body in the trachea, such as a pin, may cause

only a local sense of discomfort and dyspnœa. Bodies of moderate size (which sometimes may be unconsciously aspirated) occasion sudden onset of coughing and dyspnœa, paroxysmal exacerbations of the coughing and dyspnœa (sometimes attributable to accumulation of secretions below the obstruction), inspiratory and expiratory stridor, and the common phenomena of defective ventilation of the lungs—inspiratory depression of the suprasternal fossa and of the scorbiculus cordis, depression of the intercostal spaces, pulsus paradoxus, etc. The larynx moves but little in contrast to the marked movement that obtains in laryngeal obstruction; laryngoscopic examination reveals no disorder of the larynx; bronchoscopic examination may reveal the cause of the symptoms. The factors causing compression of the trachea from without, being usually in the neck, are quite obvious to the senses.

In bronchial obstruction, the signs and symptoms are usually unilateral, in contrast to the bilateral phenomena of tracheal obstruction. The respiratory movements of the affected side are limited, and may be completely absent if the obstruction is of high grade. If the obstruction is of moderate grade, the breath sounds are harsh and bronchovesicular in character and the fremitus is diminished; if of high grade, the breath sounds are much diminished or absent—which with normal percussion resonance is of much significance. A loud stenotic sound may be audible about the site of the obstruction. In most cases there is more or less dyspnœa—usually inspiratory. In long-standing cases some considerable difference in the circumference of the two sides may develop—due to inactivity of the affected side and compensatory emphysema of the other side. In some cases obstruction of one bronchus is followed by obstruction of the other bronchus or the trachea—as in mediastinal tumors, etc. A foreign body may lead to ulceration of the bronchial mucosa with consecutive bronchopneumonia or abscess or gangrene of the lung. Pressure from without, as by an aneurysm, may lead to retention of secretions with consecutive bronchiectasis, bronchopneumonia, and abscess or gangrene of the lung, or to collapse of the lung.

Diagnosis.—The diagnosis is usually easy. The site of bronchial obstruction may sometimes be determined by the area of diminished breath sounds, the point of maximum intensity of a stenotic murmur, by recourse to tracheoscopic or bronchoscopic examination, or, in the event of certain impervious foreign bodies, by recourse to the *x*-rays.

Prognosis.—The prognosis depends upon the cause of the obstruction. Foreign bodies, while they sometimes occasion sudden death, are also sometimes retained for months or years, and finally expectorated.

Treatment.—The treatment depends upon the cause of the obstruction. Foreign bodies may sometimes be removed by inverting the patient and exciting coughing; by direct removal with the aid of the bronchoscope; sometimes by recourse to tracheotomy or bronchotomy. Syphilis and tuberculosis are to be treated according to the basic condition. Tumors may be removed sometimes, but with much difficulty and little prospect of permanent good.

DISEASES OF THE LUNGS.

HEMOPTYSIS.

(*Pulmonary Hemorrhage; Bronchopulmonary Hemorrhage.*)

Etiology.—Hemoptysis, the spitting of blood or the discharge of blood from the respiratory tract, may result from disease of the larynx, the trachea, the bronchi (including the bronchioles), or the lung. The diseases of which it is a manifestation are: (1) Diseases of the respiratory system: (*a*) Diseases of the larynx—acute and chronic inflammation; tuberculous, syphilitic, and malignant ulceration, etc. Laryngeal hemorrhage is unusual; occasionally it is profuse, as in altered blood states, etc. (*b*) Diseases of the bronchi and trachea—bronchitis (especially the putrid and fibrinous varieties); bronchiectasis; tuberculous, syphilitic, and malignant ulceration (with erosion of a branch of the pulmonary artery); rupture of an aneurysm into the trachea or a bronchus, in which event the hemorrhage may be large and lead immediately to a fatal issue; etc. (*c*) Diseases of the lungs, especially tuberculosis (80 to 90 per cent.) of the cases, croupous pneumonia, the acute exanthemas, congestion, thrombosis, and embolism (infarction), abscess, gangrene, tumors, actinomycosis, aspergillosis, zoöparasitic infections, etc. In the early states of tuberculosis the blood usually comes from the congested mucosa of small bronchi (capillary oozing); in the later stages it is due to erosion of an artery or the rupture of an aneurysmal dilatation of a branch of the pulmonary artery. (2) Diseases of the cardiovascular system: (*a*) Advanced valvular disease (especially mitral stenosis), leading to well-marked passive congestion. The hemorrhage may be slight and recurrent, or quite free, sometimes persisting for several days. (*b*) Aneurysm of the aorta, in which, at first, the hemorrhage may be small and repeated, and due to pressure on the bronchial mucosa, or to leakage through the outer (usually fibrous) coat of the aneurysmal sac (so-called weeping of the aneurysm); or the hemorrhage may be very large (rupture of the aneurysm) and lead speedily to death. (3) Diseases of the blood—hemophilia, purpura, scurvy, leukemia, anemia, etc., and the disorganization of the blood that occurs in yellow fever, etc. (4) Diminished atmospheric pressure, as in mountain sickness, and too sudden release from caissons. (5) Arteriosclerosis and gout, so-called arthritic hemoptysis, sometimes observed in arteriosclerotic and gouty subjects with disease of the smaller vessels of the lungs. (6) Vicarious hemoptysis, sometimes observed in women at the time of menstruation or during the persistence of amenorrhœa. (7) Traumatism—injuries of the thorax, punctured wounds of the chest and lung, and rupture of a small vessel due to overexertion. (8) Neurotic hemoptysis, occasionally observed in neurotic, hysterical (especially female) subjects

who otherwise are well. Some of these subjects remain perfectly well, but some subsequently become definitely tuberculous.

Symptoms.—Rarely hemoptysis develops in persons previously in perfect health; thus, it may be the initial manifestation of tuberculosis or of an aortic aneurysm. Usually, however, symptoms of the causative disease precede the hemoptysis (which itself is only a symptom). The blood lost varies in amount from the slightest streak in a previously mucous sputum to that sufficient immediately to cause death. When small in amount the hemoptysis itself may be the only noteworthy symptom; when profuse, it is often preceded by a sense of warmth or discomfort in the chest, or of tickling or of something arising in the throat, and a saline taste in the mouth—which are followed immediately by the ejection of the blood, bright red, fluid, and frothy. The blood may be coughed up, or it may escape from the patient's mouth without obvious effort on his part. Large hemorrhages are commonly associated with mental perturbation, cardiac palpitation, marked prostration, and often syncope; immediate death may ensue, but it is rare. Fatal hemorrhage may occur into a tuberculous or other cavity without the blood being discharged externally. In other cases some of the blood effused into a cavity or bronchus is retained some time and then discharged clotted; casts of the bronchi and bronchioles may be observed. Sometimes the blood is swallowed and later vomited; or it may escape into the intestine and discolor the stools.

Physical Signs.—The physical signs, as a rule, are only those of the primary disease, with the later development of fine and coarse bubbling rales, due to the presence of blood in the bronchi and bronchioles.

Diagnosis.—The diagnosis, as a rule, is quite easy. Hemorrhage from the nose, mouth, fauces, and pharynx must be excluded (usually obvious on local examination). In hematemesis the symptoms and signs point to disease of the abdominal viscera; sensations of warmth in the epigastrium, nausea, and of faintness often precede the discharge of blood—which is vomited; the blood is usually dark in color, clotted, acid in reaction, often mixed with food, and is quite likely to appear in the stools.

Prognosis.—The prognosis depends upon the underlying disease and the amount of blood lost. Usually, the hemorrhage ceases spontaneously; but occasionally death occurs—more commonly, however, after than during the bleeding.

Treatment.—Absolute bodily and mental rest is essential. The patient should be reassured. If, as in tuberculosis, the diseased side is known, or if it is discoverable by the presence of rales anteriorly (the patient should not be disturbed for a physical examination) he may lie on the diseased side (to lessen the respiratory movements and to prevent aspiration of blood into the bronchi of the other lung). Ice may be given to the patient to suck, and an ice bag may be applied to the bleeding side, or to the precordium (to lessen cardiac excitement). Tranquillity may be favored by the hypodermic use of morphine

combined with atropine—which is also of value in restraining the cough; morphine, however, should be used rather sparingly in large hemorrhages, since by lessening the cough (benumbing sensibility), it may lead to inundation of the bronchial system with blood. Lowering of the blood pressure (the formation of a clot) should be favored by nitroglycerin, sometimes by aconite, veratrum viride, or chloroform, etc.; by purgation; by giving the minimum amount of food (cold, not hot) consistent with the patient's general condition; and by avoiding the use of cardiac stimulants, such as alcohol, strychnine, digitalis, strophanthus, caffeine, etc., even if syncope ensues. Syncope itself is a valuable adjunct in the formation of a clot. Sodium chloride on the tongue (reflex action on the vagus), and calcium chloride (30 grains, 2 grams) may prove of service. Small repeated hemorrhages may sometimes be controlled by the continued use of turpentine, or aromatic sulphuric acid. Ergot, adrenalin chloride, etc., are to be avoided, since they raise the blood pressure. Gelatin, gallic acid, lead acetate, etc., are of at least doubtful value. The hypodermic injection of normal serum has been recommended, especially in recurring hemoptyses.

CONGESTION OF THE LUNGS.

(*Hyperemia of the Lungs; Pulmonary Congestion.*)

Congestion of the lungs may be active or passive.

Active Congestion.—Active congestion of the lungs usually occurs as a secondary, symptomatic affection, but it is said occasionally to occur as a primary, independent affection. Symptomatically it may be circumscribed or diffuse, and involve one lung or both lungs. It is usually seen in association with other disorders of the lung, such as pneumonia, pleurisy, bronchitis, embolism and thrombosis, tuberculosis, etc. At times it appears to be due to the inhalation of various toxic agents, such as hot air, irritating vapors, etc., to sudden changes from cold to heat, great physical exertion, profound emotion, etc. In France it is recognized as a common independent disease, and has received the name "maladie de Woillez." Most of these cases, however, are doubtless atypical pneumonias, of which one proof is the finding of pneumococci in the sputum (believed by some writers to be attenuated pneumococci).

Symptoms.—The symptoms consist of cough, dyspnœa, and slight frothy, blood-streaked sputum, in addition to the symptoms of the associated disease. La maladie de Woillez is manifested by initial chill, fever (101° or more), cough, dyspnœa, and pain in the side of several days' duration. The physical signs are those of the first stage of pneumonia—slight impairment of percussion resonance, feeble or bronchovesicular breath sounds, increased fremitus, and fine crepitant rales.

Treatment.—The treatment is that of pneumonia.

Passive Congestion.—Two forms of passive congestion of the lungs are distinguished: (1) Mechanical, and (2) hypostatic.

Mechanical congestion results from obstruction to the return flow of blood to the left heart. It is common, therefore, in chronic mitral valvular disease, dilatation of the right ventricle, etc.; and it occurs sometimes in consequence of the pressure of mediastinal tumors. The lungs are enlarged, tough, elastic, heavy, and of a reddish-brown color (cyanotic induration), which quickly changes to a bright red color on section and exposure to the air (oxidation of the hemoglobin). Microscopically there are dilatation of the interalveolar capillaries, overgrowth of fibrous connective tissue, and in the walls of the alveoli, as well as free in the alveoli, many blood-pigmented cells (heart-disease cells of the sputum).

Hypostatic congestion occurs in many acute febrile diseases (typhoid fever, etc.), in chronic debilitating diseases, anemic, marantic, and cachetic conditions, in coma (apoplexy, and other disorders of the brain, morphine and other narcotic poisonings), in old age, etc. It results from a feeble circulation, loss of tone of the pulmonary vessels, and the influence of gravity. Usually the condition is limited to the bases of both lungs posteriorly. The affected portions of the lungs are surcharged with blood, dark red in color, œdematous, and often entirely devoid of air, whence they sink in water (hypostatic pneumonia; splenization). Occasionally in organic disease of the brain (apoplexy, etc.), the condition may be unilateral; and it is sometimes associated with definite areas of bronchopneumonia (aspiration pneumonia).

Symptoms.—The symptoms are often equivocal. Usually, however, there are cough, frothy, blood-streaked expectoration (containing pigmented, so-called heart-disease cells), dyspnœa, and more or less cyanosis. Physical examination reveals impaired percussion resonance posteriorly at the bases, increased fremitus, feeble or bronchovesicular breathing, and fine and coarse bubbling rales.

Diagnosis.—The diagnosis depends more upon the presence of a causative factor and the results of the physical examination than upon the symptoms.

Prognosis.—The prognosis depends upon the causative factor; usually amelioration of the symptoms may be brought about.

Treatment.—The underlying cause of the condition, usually organic disease of the heart, must be treated. If the symptoms are urgent, venesection should be performed; those who accept great hazards sometimes suggest tapping the right auricle—a procedure that does not appeal to me. Diffusible stimulants are often of much temporary value. The hypostatic form of the disease may often be prevented by having susceptible patients frequently turned in bed, or placed in a semiprone or upright position in bed or in a chair from time to time.

ŒDEMA OF THE LUNGS.

(Acute and Chronic Pulmonary Œdema; Recurrent Pulmonary Œdema; Acute Suffocative Pulmonary Œdema.)

Etiology.—Pulmonary œdema, the transudation into the alveoli and the interstitial tissue of the lungs, of a serous, sometimes serosanguinolent, fluid, occurs under divers circumstances: (1) As an agonal manifestation in many acute and chronic disorders, especially of the heart and bloodvessels, kidneys, lungs, blood, nervous system (brain), etc.; (2) as an acute transitory manifestation in disease of the heart, kidneys, and in certain infectious diseases; (3) as a chronic process in disease of the heart, and under circumstances akin to those that produce chronic, or passive, congestion; (4) after thoracentesis; (5) associated with inflammatory processes in the lung, especially pneumonia, tuberculosis, thrombosis, embolism, etc. (so-called inflammatory or collateral œdema; not a true œdema—rather an exudation); (6) as an apparently primary, acute, often quickly fatal disorder, developing without obvious exciting cause (acute suffocative pulmonary œdema).

The mechanism of the œdema varies in different cases. Often, as maintained by Welch, it is "due to a disproportion between the working power of the left ventricle of such a character that, the resistance remaining the same, the left heart is unable to expel in a unit of time the same quantity of blood as the right heart. Such disproportion may be brought about by paralysis or by spasm of the left ventricle," and increasing the pressure in the pulmonary circulation it leads to transudation of serum through the pulmonary capillaries. Disturbances of the innervation of the cardiopulmonary plexus by periaortitis, considerable augmentation of the vascular tension in the small circulation, and acute or rapid insufficiency of the right ventricle (Huchard), serve to explain the cases associated with angina pectoris, Hodgson's disease and other diseases of the aorta. The toxic vasomotor theory (paralysis of the pulmonary vessels) serves to explain the cases occurring in Bright's disease, in pregnancy (without valvular or other heart disease); that associated with angioneurosis; and that due to certain drugs, such as the iodides, pilocarpine, etc. Congestion by recoil (Riesman) explains those cases developing after thoracentesis, the œdema in these cases resulting from an afflux of blood to the expanding lung and a leakage of serum through the pulmonary vessels, which by reason of long compression have become changed and rendered more permeable. Increased fluidity of the blood is perhaps a factor in some cases.

Pathology.—The œdema may be limited to the bases, or it may be general and involve both lungs. The œdematous lung is bulky, pale, boggy; contains less air than normally (but does not sink in water); and on section a clear, frothy, serous or serosanguinolent fluid, of low specific gravity and containing a trace of albumin, exudes, especially on pressure.

Symptoms.—In addition to the symptoms of the primary disease, dyspnœa, cough, and expectoration are the noteworthy manifestations. The expectoration is especially characteristic, being usually abundant, thin, serous (occasionally serosanguinolent), and frothy. The dyspnœa sometimes becomes much aggravated and amounts to orthopnœa, the accessory muscles of respiration being called into activity. As a rule, there is more or less cyanosis; the pulse becomes small, rapid, and of poor tension, and the extremities become cold. There is no fever, unless bronchopneumonia or other infectious complication ensues.

Acute suffocative pulmonary œdema apparently is an independent disorder that develops suddenly, often without obvious exciting cause, and frequently leads quickly to a fatal termination, although recurring attacks are by no means uncommon. It occurs, as a rule, in arteriosclerotic, cardiac, and nephritic subjects, and is characterized by the sudden development, often at night, of attacks of extreme dyspnœa (orthopnœa), cyanosis (pallid lividity), cough, expectoration of a thin, frothy, serous, albuminous (occasionally blood-streaked) fluid, and marked prostration (with sweating, etc.). The fluid often drools from the patient's mouth without effort on his part; I have seen twelve ounces of such fluid expelled within a few hours. The attack may result in death within ten or fifteen minutes, the patient being virtually drowned in his own fluids; or it may subside rather soon (within a half hour or thereabout), or it may last from three to twelve or more hours. The condition of the heart varies during the attacks; in one patient that I observed the right heart seemed to be considerably dilated during the attack.

Physical Signs.—Physical signs consist of general impairment of the percussion resonance, or scattered foci of dulness and intervening areas of vesiculotympanitic sound (relaxation of the lung tissue), feeble, harsh, or obscured vesicular breathing, and numerous fine and coarse bubbling rales (localized or distributed generally throughout both lungs).

Diagnosis.—The diagnosis is easy; the symptoms and physical signs can scarcely be mistaken for anything else. An associated bronchopneumonia should not be overlooked.

Prognosis.—The prognosis is grave, especially in view of the associated phenomena, of which the œdema is often a terminal manifestation.

Treatment.—The treatment, as a rule, is that of the disease of which the œdema is one manifestation. A hypodermic injection of morphine ($\frac{1}{4}$ grain, 0.015 gram) and atropine ($\frac{1}{100}$ grain, 0.0006 gram) will usually almost immediately relieve the severe symptoms; I have seen it render the greatest service (apparently save life) in the acute suffocative type of the disease. The injection may be repeated, if the circumstances demand it; repeated injections of atropine are often of value for a day or two. In addition, diffusible stimulants are indicated—camphor, strychnine, ether, whiskey, etc., hypodermicly; strophanthus, coffee, brandy, whiskey, champagne, etc., by the mouth. Venesection is sometimes a life-saving procedure, and should be resorted

to when the patient's condition is grave (extreme cyanosis, etc.). Counterirritation (dry or wet cups, mustard poultices, etc.) may prove of service. Drastic purgatives are often given in the hope of relieving the system of fluid, but their action is too severe to recommend their use while the patient is seriously ill. Nor does the use of emetics to assist in the expulsion of the fluid commend itself to me. Since the disorder is often a terminal manifestation, treatment in these cases may not prove of avail.

PULMONARY EMBOLISM AND THROMBOSIS.

(*Pulmonary Infarction; Hemorrhagic Infarction of the Lung; Pulmonary Apoplexy.*)

Etiology.—Primary thrombosis of the pulmonary artery or its branches is an unusual event, but it may result from slowing of the circulation, impoverishment of the blood, and disease of the arterial wall, such as may occur in pneumonia (and other infections), arteriosclerosis, compression of the vesse by a tumor, etc. Pulmonary embolism results from thrombotic processes in the right side of the heart or in the systemic veins, especially the femoral veins, the veins of the uterus (after parturition), the sinuses of the brain, the veins of the abdomen, liver, etc. It is common in the sequence of mitral valve disease, especially stenosis, in which passive congestion of the lungs and dilatation of the right side of the heart have supervened, the thrombi developing in the auricle or amidst the columnæ carneæ. The foundation of the embolus may also be a newgrowth that has invaded a vein, a phlebolith, fat set free by a fracture of a long bone, rupture of the liver, rupture of the subcutaneous adipose tissue, air bubbles, etc. The embolus, as well as the thrombus, may be infectious or non-infectious.

Pathology.—The embolus or thrombus may involve the main branch of the pulmonary artery, one or several of the medium-sized branches, or the smallest branches. The blocking of the vessel is usually, but not always, followed by infarction; sometimes not, because death occurs almost immediately; in other cases, because the obstruction is not complete, or the collateral circulation is sufficient, or the bronchial arteries maintain the circulation. Stasis of the pulmonary circulation seems to be an important if not essential element in the process of infarction. The infarct may be single, though they are usually multiple; they are more common in the lower lobe on the right side; they vary in size from a marble to an orange; they consist of pyramidal or wedge-shaped masses, with the base toward the periphery, dark red in color, firm, and devoid of air. Microscopicly they are found to consist of blood filling the air vesicles and infiltrating the lung tissue. The thrombus or embolus often escapes detection. The overlying pleura is often covered with a fibrinous exudation, through which small foci of hemorrhage may be observed. Small infarcts may be absorbed, or the blood may be removed by reëstablishment of the circulation;

large infarcts leave behind a puckered scar. Should the embolus be infective, pneumonia, abscess or gangrene of the lung, pleuritis or pyopneumothorax, etc., may result.

Symptoms.—Complete obstruction of a main branch of the pulmonary artery is often followed by immediate death; in other cases, after a few moments of a feeling of extreme constriction within the chest, dyspnœa, and terror, the patient loses consciousness, the pupils dilate, the face becomes livid, the pulse rapid and irregular, perspiration breaks out, and death occurs within an hour. In less severe cases, that is, in cases in which the obstruction is not complete or the obstructed vessel is small, the chief symptoms consist of sudden pain and a sense of constriction within the chest, dyspnœa (labored, sometimes Cheyne-Stokes breathing), hemoptysis, and more or less collapse. Bloody, dark-red, non-aërated expectoration is quite significant of infarction. A typical "stitch in the side" sometimes ensues, and is referable to a concomitant pleurisy; it is usually associated with local tenderness and a friction rub. Fever does not occur, unless the embolus is infective; in this event, to the general manifestations of septicopyemia are added the local signs of pulmonary abscess or gangrene. Occasionally small infarcts develop without noteworthy symptoms.

Physical Signs.—There are no characteristic physical signs. Small peripheral and central infarcts usually occasion no physical signs whatever; but larger infarcts that reach the pleura may occasion dulness on percussion, harsh bronchial or bronchovesicular breath sounds, and crepitant or crackling rales. Should there be a local pleuritis, the breath sounds may be feeble or inaudible, and frictions may develop.

Diagnosis.—The diagnosis may be exceedingly easy, as in a chronic heart subject in whom the symptoms develop typically; in other cases, it may be quite difficult—in case of sudden death. In these cases, the diagnosis is facilitated by the presence of a causative factor, and the absence of definite causes for thrombosis or embolism of the coronary artery, cerebral hemorrhage, rupture of an aneurysm, and other causes of sudden death.

Prognosis.—The prognosis, as a rule, is unfavorable. If death does not occur soon, the infarction, nevertheless, points to weakness of the right ventricle, a primary thrombus somewhere in the body, or other serious disorder. Occasionally the infarct is absorbed and the patient recovers temporarily, at least. Recurrences are rather common.

Treatment.—Prophylaxis is of the utmost importance. Patients in whom venous thrombi exist, or who are ill with diseases known to favor the development of such thrombi, should be carefully nursed, protected from sudden movements that might dislodge the thrombus, and well fed. At the time of the development of the embolism, morphine and atropine are usually necessary to relieve pain; if the patient does not die soon, recourse should be had to stimulants—camphor, strychnine, ammonia, oxygen, etc. Later the primary disorder requires attention.

ATELECTASIS AND COLLAPSE OF THE LUNG.

(*Apneumatosis; Compression of the Lung.*)

Atelectasis and collapse of the lung are much-related conditions: atelectasis properly is a condition found in infants in which the lungs or portions of the lungs have not expanded; collapse occurs in older persons and is a reversion to this condition after expansion has occurred. Both terms, however, are commonly used interchangeably. Congenital and acquired forms of the condition are usually distinguished.

Etiology.—*Congenital atelectasis* results from inefficient action of the respiratory muscles (associated usually with general feebleness of the infant); defective innervation resulting from a congenital defect or from pressure on the fœtal head by the forceps or a contracted pelvis; obstruction of a bronchus by mucus or meconium, or by pressure from without, as by enlarged lymph nodes; or by pressure on the lung by enlarged abdominal organs, tympanites, ascites, etc.

Acquired atelectasis, or collapse, of the lung results from: (1) Obstruction of a bronchus by mucopus, foreign bodies, newgrowths, or other products of inflammation. It is found most commonly in association with bronchopneumonia. When the obstruction is complete, air can neither enter nor leave the involved portion of the lung, and absorption of the air by the blood takes place. (2) Compression of a bronchus by a pleural effusion, hydrothorax, pneumothorax, pyothorax, pericardial effusion, extreme cardiac hypertrophy, aneurysm of the arch of the aorta, newgrowths of the thorax and mediastinum, kyphoscoliosis, and pressure from below the diaphragm by enlarged viscera, tympanites, ascites, etc. (3) Feeble respiratory efforts, such as occur in debilitated and asthenic conditions generally—cachectic and marantic states, typhoid fever, coma, prolonged dorsal decubitus, paralysis of the pneumogastric nerves, rachitis, pleural adhesions, kyphoscoliosis, etc.

Pathology.—Atelectasis most commonly involves the lower portions of the lungs; it may occur in scattered foci (obstruction within the bronchi,) or it may implicate an entire lobe (compression from without). The collapsed portion of the lung is reduced in size, purplish red in color, firm, airless, and sinks in water; sometimes there is serous infiltration (splenization, carnification). In the congenital variety the atelectatic portions of the lung may gradually expand, or they may remain unexpanded and become the seat of fibrous thickening—hypoplasia or aplasia of the lung. The condition is often associated with congenital anomalies of the circulation (patulous ductus Botalli, foramen ovale, etc.).

Symptoms.—*Congenital atelectasis* is manifested by feeble and superficial respiration, dyspnœa, cyanosis, cold extremities, feeble heart action, drowsiness, sometimes muscular twitchings and convulsions (carbon dioxide poisoning). The physical signs consist of deficient

expansion of the chest, retraction of the intercostal spaces, and defective inspiratory murmur over the atelectatic portions of the lung.

The symptoms of *acquired atelectasis* are often obscured by those of the primary disorder, commonly bronchopneumonia, and the collapse often is not diagnosticated. In other cases, dyspnœa, cyanosis, feeble heart action, cold extremities, etc., develop, but it is difficult to say whether these are not due as much to the primary disorder as to a supposed collapse of the lung. In cases associated with kyphoscoliosis, cyanosis, dyspnœa, and palpitation of the heart are usually marked, and in consequence of the obstruction in the pulmonary circulation considerable hypertrophy and dilatation of the right ventricle ensue; ultimately lack of compensation supervenes.

Physical Signs.—When associated with bronchopneumonia there are often no noteworthy physical signs in addition to those occasioned by the bronchopneumonia; sometimes to the evidences of localized foci of solidification are added those of intervening (or surrounding) foci of inflation. When the collapsed area is large (compression of a bronchus from without), there is restricted movement at the base of the chest (or over the collapsed area), with increased expansion elsewhere, impaired percussion resonance, diminished fremitus, and feeble breath sounds; occasionally, when the air has access to the main bronchus, there may be increased fremitus and bronchovesicular breathing. Crackling rales are not uncommon; frequently they disappear after repeated deep inspirations.

Diagnosis.—The disorder is often overlooked when the foci are small; when large, the physical signs serve for their recognition. Changes in the dulness, breath sounds, and rales after several deep breaths or a change of posture (for several hours) serve to exclude many other disorders, such as pneumonia, pleuritis, thrombosis and embolism, etc.

Prognosis.—The prognosis depends upon the associated conditions. Sudden death may occur in kyphoscoliosis, and in bronchopneumonia in which change of posture, etc., does not lead to inflation of the collapsed areas.

Treatment.—The treatment in general is that of the primary disorder. In addition, measures that tend to prevent the development of the atelectasis should be employed. Thus, patients should be prevented from lying in the one position for a long time, and deep inspirations should be insisted upon from time to time. Cold affusions are of service in preventing the disorder, and of value when it has developed. Diffusible cardiac stimulants (camphor, ether, strychnine, ammonium preparations, etc.) are valuable, and so also sometimes are oxygen, compressed air, etc. In congenital atelectasis, forcible inflation has been productive of good results.

BRONCHOPNEUMONIA.

(*Lobular Pneumonia; Capillary Bronchitis.*)

Bronchopneumonia is a bacterial infection of the lungs characterized by inflammation of the terminal bronchioles and the related alveoli that collectively make up a lobule. It is not distinguishable from capillary bronchitis.

Etiology.—Bronchopneumonia is most common in children under five years of age (75 per cent. of the cases) and in the aged; but adults are by no means exempt. It may occur at any season of the year, but it is especially prevalent during the winter and spring. Poor hygienic surroundings, malnutrition, etc., act as predisposing factors. The exciting causes comprise the pneumococcus, Friedländer's pneumobacillus, streptococci, staphylococci, the influenza bacillus, the typhoid bacillus, the tubercle bacillus, etc. The majority of cases are mixed infections.

Primary and secondary cases may be distinguished. The primary cases are relatively uncommon; they occur particularly in previously healthy children under two years of age, and are usually due to the pneumococcus. The secondary cases are of several types: (1) Those complicating or following the infectious diseases, especially measles, pertussis, diphtheria, scarlatina, influenza, typhoid fever, etc., as well as other diseases attended by bronchitis. This group is especially common in children. In some cases of tuberculosis of the lungs of the ordinary chronic ulcerative type, bronchopneumonia (sometimes terminal) may develop, and is due frequently to the pneumococcus, although the lesions may be due to mixed infection. (2) Those complicating chronic debilitating disorders, such as nephritis, diabetes, chronic cardiac disease, prolonged confinement to bed (as in fracture of the femur), etc. This type is especially common in adults, particularly the aged, and is often responsible for the final fatal issue, constituting a so-called terminal infection. (3) Aspiration pneumonia, which occurs when the sensibility of the larynx has become benumbed, as in stuporous and comatose states, such as uremic or diabetic coma, apoplexy, ether and chloroform anesthesia, or operations on the larynx or trachea, etc.—in which circumstances bacteria-laden particles of food, or saliva, etc., are drawn into the bronchi and ultimately set up inflammation. The pneumonia that may follow non-fatal drowning, the inhalation of irritating vapors, gases, etc., is perhaps of the same nature. (4) Perhaps in some cases the infection is carried to the lungs by the blood stream or the lymph stream.

Pathology.—The lesions consist of scattered foci of solidification, varying in size from that of a pinhead to a cherry or larger; the foci are bilateral and usually most marked in the lower lobes. On section they consist of irregular, dark, reddish-brown (later, grayish or yellowish), firm, airless areas, in the centre of which a bronchiole can

often be detected; it is usually filled with mucopus, and its epithelium is swollen and reddened. Surrounding vesicles are often atelectatic; perhaps in some cases the atelectasis precedes the inflammatory phenomena. Microscopically, the bronchioles show the ordinary vascular and exudative lesions of inflammation, especially cellular infiltration, and proliferation and desquamation of the epithelium, which, with the mucopus, obstructs the bronchioles; the related alveoli are more or less filled with leukocytes, some erythrocytic cellular exudation, proliferated and desquamated alveolar epithelium, and some fibrin formation—which is most marked in the pneumococcic cases (although always less than in so-called lobar pneumonia), and less in pyococcic and influenzal cases, etc.; the alveolar walls are swollen and infiltrated and show the ordinary lesions of inflammation. These disseminated foci of peribronchial solidification and atelectasis, and embracing areas of more or less well aërated lung, constitute the common type; in some cases bronchitis and bronchiolitis with comparatively little (although always some) alveolar involvement are the conspicuous lesions; whereas in other cases the lesions spread from the primary foci directly from one alveolus to adjacent alveoli or through the lymphatics until more or less of the lobe is involved (pseudolobar form); but intervening areas of more or less congested pulmonary tissue separate the solidified lobules. The bronchopneumonic lesions may resolve (autolysis, fatty degeneration, etc.) and become absorbed, perhaps in part expectorated; or if death does not ensue from exhaustion and toxemia, pulmonary abscess, gangrene, or fibrosis may ensue.

Symptoms.—In primary bronchopneumonia the onset may be sudden, following slight exposure (as in ordinary lobar pneumonia); there may be a chill (or chilliness, or perhaps a convulsion in young children), rapid rise of temperature, cough, and dyspnœa. In some cases the pulmonary symptoms are overshadowed by the general, or perhaps by unusual cerebral manifestations (stupor, coma, delirium, etc.). Secondary bronchopneumonia, following, for instance, one of the acute infections of children with bronchitis, is usually of insidious onset; it is characterized by exaggeration of the already existing fever, increased cough and dyspnœa, and cyanosis, any or all of which are indicative of bronchopneumonia in a susceptible bronchitic subject; rarely the onset is sudden. The fully developed disease is characterized by irregular fever (103° to 105°), rapid, feeble pulse (140 to 160 per minute in children), hard, ineffectual, and distressing cough, dyspnœa (60 to 80 or more respirations per minute in children), activity of the accessory muscles of respiration, inspiratory retraction of the lower part of the chest, cyanosis, marked prostration, stupor (and other so-called meningitic manifestations), leukocytosis, etc.

Physical Signs.—The physical signs in the early stages and in some cases throughout the course of the disease, consist of harsh bronchovesicular (puerile) breath sounds and fine crackling and sibilant rales throughout both lungs; that is, signs of solidification are altogether

wanting (the capillary bronchitis of some writers). In other cases there are in addition scattered foci of impaired resonance, sometimes of more or less well-marked dulness, and increased tactile and local fremitus, especially at the bases posteriorly; in the event of widespread involvement of a lobe more or less outspoken bronchial (tubular) breath sounds may be heard.

Similar symptoms and physical signs develop in the event of aspiration or deglutition pneumonia. The bronchopneumonia that develops in adults in debilitated and cachectic states is often extremely insidious in onset; symptoms often are markedly in abeyance; there may be only a little dyspnœa, or perhaps cyanosis, and the disease may be overlooked unless the chest be examined.

Diagnosis.—The diagnosis of the common secondary bronchopneumonia of children should be based upon the primary disorder and the advent of increased fever, cough, dyspnœa, and cyanosis; the physical signs of solidification often may be entirely absent. Acute bronchitis may be differentiated by the low fever, less dyspnœa, and less cyanosis; there is no clinical and little anatomical difference between capillary bronchitis and bronchopneumonia. Lobar pneumonia may be distinguished by its comparative rarity in infants, and its characteristic mode of onset, physical signs, and course. Efforts should always be made to determine the etiological factor in each case by examining the sputum.

Prognosis.—The course of bronchopneumonia may rarely be acute, lasting about a week or ten days; usually it lasts from three to six or eight weeks or more. Death may ensue from divers complications, toxemia, or asthenia; recovery ensues by lysis. The prognosis should always be guarded, especially in the very young and the aged; it is especially bad in the cases following measles and pertussis in debilitated and rickety children, in aspiration pneumonias, and generally when there is marked cyanosis, dyspnœa, toxemia, and prostration.

Treatment.—The treatment, in general, is that of the primary disorder, or of acute bronchitis, or of croupous pneumonia. The most important measure, especially in young children, consists of an abundant supply of fresh, cool air, which, ordinarily, is best obtained by open windows or by roof gardens at hospitals, etc. Fresh, cool air stimulates the vasomotor system and the heart, relieves the cyanosis, and lessens the toxemia and its cerebral effects—and is much superior to canned oxygen in a warm, ill-ventilated room. The patient should be well protected by a sufficiency of, but not too many, bedclothes (remembering the fever); the feet should always be kept warm and the head cool. The patient should not be distressed by poultices, blisters, and other counter-irritants. If the physician must contend against local prejudices, let him apply a loose-fitting cotton jacket (which may take the place of other clothing). The diet should be fluid. An abundance of cold water should be given to drink. The bowels should be opened thoroughly—castor oil or magnesia in children; calomel or blue mass and

a saline cathartic in adults. The use of water externally and internally as recommended in croupous pneumonia, is serviceable; in children a warm bath is often better; in adults, as well as in children, the desired result of hydrotherapy is reaction and stimulation, and not reduction of temperature. Diffusible stimulants, such as whiskey and brandy, strychnine, ammonia, and camphor, comprise the really efficacious remedies; coal-tar products and all depressing drugs should be absolutely avoided. Restlessness, pain, excessive cough, etc., suggest the use of bromides, Dover's powder, paregoric, etc., all of which should be used with caution in the young and the aged. The other therapeutic indications are similar to those mentioned under bronchitis and croupous pneumonia.

CHRONIC INTERSTITIAL PNEUMONITIS.

(*Chronic Fibroid Pneumonia; Fibroid Induration of the Lung; Sclerosis of the Lung, Fibroid Phthisis.*)

Etiology.—Chronic interstitial pneumonitis may occur as a primary or a secondary process. The primary process is due to syphilis or to the inhalation of irritating dust (pneumokoniosis). The secondary process may follow croupous pneumonia, bronchopneumonia, tuberculosis, collapse of the lung (due to an aneurysm, a tumor, etc.), chronic pleuritis, etc. A local fibrosis is common in association with healed foci of tuberculosis, especially at the apex, gummas, pleural thickenings, gangrene, tumors, hydatid cysts, etc.

Pathology.—The lesion may be localized or diffuse, unilateral or bilateral. They consist of proliferation of the fibrous connective tissue, which begins in the interlobar or interlobular septa or about the bronchioles, depending upon the variety of the disease. In the form that follows croupous pneumonia the exudation, instead of undergoing autolysis, becomes organized; fibroblasts invade it and ultimately lead to a fibrillar thickening of the alveolar walls and obliteration of many of the alveoli. The affected portion of the lung is contracted, smooth, firm, dense, homogeneous, semitranslucent, grayish white in color, and almost if not quite airless. Often an entire lobe is involved, in which event it may be much reduced in size and lie against the spine as an irregular, dense mass. In the form that follows bronchopneumonia, the fibrosis begins about the bronchi; the alveolar walls become much thickened, and small foci, lobular masses, smooth, firm, grayish in color, are found scattered throughout a lobe or an entire lung. On section these areas usually reveal a central bronchiole. From contraction of the newly formed fibrous tissue bronchiectasis not infrequently results. The pleurogenous form may follow prolonged compression of a lung by a pleural exudate; in other cases bands of fibrous connective tissue extend from the pleura (sometimes only the interlobar pleura) a variable distance into the lung, and by cicatrization lead to compression and atrophy of the lung tissue, and to bronchiectasis. At times the lung

consists virtually of a series of bronchiectatic cavities, in the walls of which aneurysmal dilatations of the pulmonary artery are not uncommon. Bronchitis (fibrous peribronchitis) is a part of the general process of indurative pneumonitis. Tuberculosis may ensue. The uninvolved portion of the lung, or the opposite lung, is commonly the seat of compensatory inflation. The heart is often displaced, retracted toward the diseased side; the right ventricle is usually hypertrophied.

Symptoms.—The symptoms are often merged with those of the primary disorder—croupous pneumonia, bronchopneumonia, pleuritis, etc. The patient does not fully recover—slight cough, mucopurulent expectoration, dyspnœa, and perhaps a little fever persist for some time. Gradually the symptoms become only those of chronic bronchitis; later, usually those of bronchiectasis, which with the physical signs of retraction of the chest are quite characteristic of the disease.

Physical Signs.—The chest is depressed over the affected lung or part of the lung; the interspaces are obliterated; the ribs may overlap; the shoulder is depressed; the nipple and the scapula are nearer the median line; scoliosis even may develop. The inspiratory expansion of the chest is restricted; that of the unaffected lung may be increased. The tactile and the vocal fremitus are increased. On percussion there is dulness or a vesiculotympanitic sound (bronchiectatic cavity). On auscultation there is harsh bronchovesicular breathing, or the breath sounds are feeble (thickened pleura, etc.); and crackling, sibilant, sonorous, and bubbling rales and sometimes pleural frictions may be heard. Retraction of the left lung by uncovering the heart causes an increase in the area of cardiac dulness (some of the increase is due to hypertrophy and dilatation of the right ventricle), increased visible and palpable pulsation of the heart and adjacent vessels (pulmonary artery and the aorta), palpable closure of the pulmonary valve, increase of the tympany of Traube's semilunar space; etc. Retraction of the right lung causes displacement of the heart to the right, elevation of the liver, etc.

Diagnosis.—Usually for some time the diagnosis is that of the primary disorder, or of bronchitis. With the development of retraction of the chest the diagnosis becomes quite obvious. Tuberculosis should be excluded.

Prognosis.—Although the disease is incurable, the patient may live many years and enjoy tolerably good health. Exacerbations of the bronchitis are not uncommon. Death usually results from progressive toxemia (bronchiectasis), failure of the right heart, or some intercurrent infection (bronchopneumonia, etc.).

Treatment.—The treatment is that of chronic bronchitis and bronchiectasis. A nutritious diet and the best hygienic and climatic conditions are of prime importance.

PNEUMOKONIOSIS.

(*Anthracosis; Chalicosis; Siderosis.*)

Etiology.—Pneumokoniosis is a term applied to the fibroid and other changes in the lungs that result from the inhalation of various kinds of dust. The condition is essentially an occupation disease, and is seen especially in those constantly exposed to dust-laden atmosphere. Different varieties of the disease are distinguished, depending upon the nature of the dust inhaled: (1) Anthracosis, that due to the inhalation of carbonaceous pigment, coal dust, soot, charcoal, etc. (coal-miner's disease, or phthisis); (2) chalicosis, that due to the inhalation of silicious dust, such as sandstone, quartz, etc. (stone-cutter's phthisis, millstone maker's phthisis, grinder's and potter's rot or asthma); (3) siderosis, that due to the inhalation of mineral dust, especially iron oxide, brass, bronze, etc.; (4) that due to the inhalation of particles of wool, flax, cotton, etc.; and (5) that due to the inhalation of grain dust, cereals, etc. (miller's phthisis). Associated bacterial infection is doubtless of some significance.

Pathology.—Small amounts of dust being inhaled are absorbed by phagocytes (leukocytes and other cells), which then are carried upward by the ciliated epithelium and expectorated. When larger amounts are inhaled, some penetrates to the mucosa of the small bronchi, where it is attacked (engulfed) by the normal scavengers of the respiratory tract, the mucous, and the alveolar cells. When the amount of the dust is excessive some of it reaches the alveoli, and some of it penetrates the mucosa and reaches the lymph stream. In this event it may be carried to the lymph nodes surrounding the bronchi and bloodvessels, to the interlobular septa beneath the pleura, and to the substernal, bronchial, and tracheal lymph nodes. Rarely the dust particles gain the general circulation, and may be found in the liver, spleen, kidney, mesenteric and retroperitoneal lymph nodes, etc.—cases in which the pigmented bronchial nodes have formed an intimate connection with the pulmonary veins and permit the passage of dust-laden cells into the veins. In the large majority of cases there is no reaction to the deposit of these foreign bodies in the lung, and the lungs show no noteworthy deviation from the normal, aside from variations in color depending upon the special pigment deposited (black, coal dust; whitish or grayish, stone dust; yellowish, reddish, or black, iron dust). In some cases, however, reaction does take place: this consists of the proliferation of fibrous connective tissue beginning in the peribronchial lymph nodes, to which it may remain localized—in which event the fibroid nodules appear as firm, hard, circumscribed masses, varying in size from a millet seed to a cherry, scattered throughout both lungs, and on section revealing in the centre a bronchiole. In other cases the process is more diffuse, and involves almost, if not quite, an entire lobe. In these cases there is also thickening of the alveolar walls and of the interlobular and perivascular

tissue, so that the affected portion of the lung is contracted, dense, firm, and almost airless; in some areas the bronchi and alveoli may be completely obliterated. An important element in the process is the associated bronchitis (which causes most of the symptoms) and emphysema; bronchiectatic cavities are common; in other cases necrosis of affected areas takes place and leads to the formation of cavities with irregular and roughened walls, traversed by fibrous bands, communicating or not with a bronchus, and containing thick, dark, sometimes blood-stained fluid. Tuberculosis is a common sequel.

Symptoms.—The symptoms are those of chronic bronchitis and emphysema—cough, dyspnœa (especially on exertion), and expectoration, which, however, do not come on until the subject has worked in the dust for a number of years. The expectoration is usually profuse, mucopurulent, and blackish in anthracosis; in chalicosis one may detect microscopically angular crystalloid particles of silica; and in siderosis, yellowish or reddish particles of iron oxide. In the course of time the general health begins to fail, and the patient may become much emaciated.

Physical Signs.—The physical signs are those of chronic bronchitis and emphysema; later, those of chronic induration of the lung, and cavity formation.

Diagnosis.—The diagnosis is usually easy, if the patient's occupation is taken into account. Cavities should not be misinterpreted as tuberculous necessarily; tubercle bacilli may be absent throughout the entire course of the disease.

Prognosis.—The prognosis as regards life expectancy is rather good. The patient may survive many years. Death often results from secondary tuberculous or other infection (bronchopneumonia, etc.). The development of bronchiectasis and excavation renders the prognosis unfavorable.

Treatment.—The treatment is that of chronic bronchitis and emphysema. The patient should give up his occupation if possible; if this be impossible, he should spend considerable time in the open air.

EMPHYSEMA.

Emphysema is a chronic disease of the lungs characterized by permanent overdistention of the air vesicles and atrophy of the vesicular walls.

Etiology.—True (large-lunged, pseudohypertrophic) emphysema is peculiarly a disease of adult life, although well-marked examples are occasionally seen in children. It is most common in the male sex. In some cases it seems to be predisposed to by heredity (hereditary weakness of the pulmonary elastica and consequent deficiency of contractile power). It is especially common in those whose occupation requires prolonged increase of intrapulmonary pressure with a closed glottis and muscular effort, such as players on cornets and other wind instruments, glassblowers, and those that lift or move heavy bodies (pianos,

etc.). Advancing age is a predisposing factor, but this usually results in so-called senile (small lunged) emphysema. The disease is especially common as a secondary process in diseases attended with violent coughing spells and difficulty in expiration, such as pertussis, chronic bronchitis, asthma, etc. In these there is always obstruction to the exit of air through the bronchioles and consequent increased intra-alveolar pressure, which becomes markedly augmented during the violent expirating efforts of coughing.

Pathology.—Both lungs are obviously enlarged, filling an enlarged thorax, depressing the diaphragm, and overlapping the heart; they are pale and anemic (aside from the common anthracosis), and characteristically light and downy to the touch; that is, despite the obvious increase in size, there is a lack of pulmonary tissue. Larger or smaller bullæ (coalescence of several alveoli) may be seen at different parts of the lungs, but especially at the free margins, anteriorly and basally (which become rounded), and the apices (less well supported than elsewhere), and that portion in the vertebral fossæ. The conspicuous lesions consist of dilatation of the alveoli and infundibula; atrophy of the interalveolar walls, including the elastica; consecutive thrombosis and obliteration of the interalveolar capillaries and also the lymphatics; fatty and other degenerative processes of the epithelial lining of the alveoli; and the eventual establishment of abnormal communications between adjacent dilated alveoli. The bronchi and bronchioles show the common lesions of chronic inflammation—being thickened, indurated, dilated, and distorted. There is more or less thickening and sclerosis of the pulmonary artery, which together with restriction of the arterial tree from capillary thrombosis, leads to hypertrophy of the right ventricle; the increased tension in the pulmonary artery is somewhat relieved by normal communications between the pulmonary artery and the pulmonary and bronchial veins—but this leads to increased cyanosis, since the blood thus diverted does not become well aërated.

There is still doubt regarding the real nature of the process. Although some observers still hold that it results from inspiratory distention or from primary degenerative changes of the alveolar walls, the prevailing view is that it is due to expiratory difficulties and consequent increase in intra-alveolar pressure—which becomes especially significant etiologically when acting upon lungs reduced in resisting power by hereditary influences, chronic inflammation (bronchitis), senility, etc. At the time of greatest intra-alveolar pressure (at the acme of coughing, straining, etc.) the lungs are inflated to the maximum, the glottis is closed, and the thorax fixed, so that air is forced into parts of the lungs normally slightly expanded, that is, the apices and the anterior margins, which being also ill supported, feel the strain most and reveal the most marked lesions. In consequence of repeated insults, the pulmonary tissue gradually loses its elasticity, and the lungs gradually become permanently almost, if not quite, maximally distended; and the chest, permanently in the condition of inspiratory expansion, assumes the characteristic barrel-shape. Some

observers have postulated primary ossifying changes in the costal cartilages.

Symptoms.—The significant symptoms of emphysema are progressive dyspnœa and cyanosis. At first these are noticeable only on severe exertion, but as the disease advances they develop on the slightest exertion and then become permanent. The dyspnœa is chiefly expiratory, and is increased by mental excitement and bodily exertion. The respirations are increased in frequency (20 to 30), and ultimately orthopnœa supervenes. Cyanosis is an early and prominent symptom, apparent first in the lips, nose, ears, and extremities, but becoming more marked as the disease advances; it is often of a strikingly high grade, without notable discomfort to the patient. It is due in part to the disease of the lungs, in part also to failing cardiac compensation (dilatation ensuing upon the hypertrophy due to the pulmonary disease). Usually there is a co-existing bronchitis, which becomes aggravated in the winter, and occasions cough and mucopurulent expectoration; asthmatic attacks are common. So long as the hypertrophy of the right ventricle continues efficient the patient is tolerably comfortable; but eventually dilatation and failure of compensation ensue, whereupon increased dyspnœa and cyanosis, pulmonary œdema, and signs of general venous engorgement are likely to supervene.

Physical Signs.—The aspect of the patient is often quite characteristic: if the disease is at all advanced he is markedly cyanotic and more or less dyspnœic, his eyeballs bulge, his head is thrown back and the chest forward, and the accessory muscles of respiration are active. The chest is characteristically barrel-shaped—shortened and increased anteroposteriorly; the neck is short and thick; the supraclavicular fossæ may bulge; the sternum is prominent, the costal angle increased; the shoulders are raised and rounded, the ribs almost horizontal, and the intercostal spaces widened and depressed. The movements of the chest are vertical rather than expansile; the inspiratory movements are shortened, the expiratory prolonged; respiration is carried on largely by the diaphragm and the abdominal muscles, although the excursions of the diaphragm are restricted (obvious by the use of the *x*-rays and in the limitation or absence of Litten's diaphragm sign). A line of distended capillaries and venules around the chest often marks the attachment of the diaphragm. The percussion note is hyperresonant or vesiculotympanitic; in some places dull (imperfect resonance of the thorax due to tension or ossifying costal cartilages). The increased volume of the lungs is revealed in percussion resonance to the seventh rib or below in the mamillary line and to the first or second lumbar spine posteriorly, by reduction in size and eventual obliteration of the superficial cardiac dulness, encroachment of resonance on the hepatic and splenic dulness, pulmonary resonance in Traube's semilunar space, and little or no respiratory movement of the lower limits of pulmonary resonance (in Gerhardt's space and at the cardiopulmonary borders). The breath sounds are weakened, sometimes almost inaudible, and

the expiration is much prolonged in uncomplicated emphysema; with complicating bronchitis, the breath sounds are usually harsh and broncho-vesicular in character, and rales may be heard. Adventitious sounds may also be due to shifting of air in emphysematous bullæ, pulmonary œdema, muscular contractions, etc. The cardiac apex is usually not to be seen or felt; a common palpable pulsation in the epigastrium results from downward displacement of the heart and hypertrophy of the right ventricle. The heart sounds are weak and indistinct, and there may be a systolic murmur (cardiopulmonary; relative tricuspid or mitral insufficiency).

Diagnosis.—The disease is readily recognized from the history and the physical signs, although minor grades are often overlooked or misinterpreted. The cyanosis, dyspnœa, and a possible systolic murmur should not be mistaken for primary cardiac disease; the other physical signs are unequivocal. Pneumothorax is sometimes confused, on account of the clear vesiculotympanitic percussion rate.

Senile Emphysema—In contradistinction to the so-called large lunged emphysema, a small lunged (atrophic or senile) emphysema is often encountered in elderly people. This is a primary atrophy of the pulmonary tissues, part of the general atrophy of senility. It is attended by some dyspnœa, but not much cyanosis; there may or may not be cough, but if present it is not marked. The chest is small and flat; the normal obliquity of the ribs is perhaps accentuated; the musculature is atrophic; the normal type of breathing is maintained; the percussion note is hyperresonant, and the breath sounds are weak, unless there be associated bronchitis.

Compensatory Inflation (Vicarious Emphysema).—In local disease of the lungs, such as pneumonia, bronchopneumonia, more or less quiescent tuberculosis, chronic fibrosis, pleural adhesions, pleural effusion, parts of the lung adjacent to the diseased foci, or a lobe, or the opposite lung, takes on a compensatory process, becomes distended—so-called compensatory emphysema (preferably inflation or distention). This is at first a purely physiological process—enlargement of the alveoli, but no atrophy of the walls; it is believed to be inspiratory rather than expiratory in development; it may be temporary, and, as in pneumonia or pleural effusion, subside with the subsidence of the primary disorder, but if long continued, true emphysema may ensue.

Acute Vesicular Emphysema.—Acute vesicular emphysema (or distention) sometimes ensues when violent inspiratory efforts have been made in conditions of bronchial obstruction, especially in bronchitis of the fine tubes, asthma, pertussis, aortic aneurysm, etc. The percussion note is hyperresonant and more widely distributed than normally, the expiration is prolonged, and there are many sonorous and sibilant rales. It is readily distinguished by its usually acute onset, associated phenomena, and common subsidence.

Interstitial Emphysema.—Interstitial (interalveolar, interlobular, or subpleural) emphysema is a condition characterized by the presence

of air in the interstices of the lung, where it forms larger or smaller bullæ. It may be due to trauma (fracture of the ribs, punctured wounds of the chest, tracheotomy, etc.); rupture of the air vesicles from overdistention (severe paroxysmal coughing, straining of parturition or defecation, etc.); it may follow pneumothorax, subcutaneous emphysema of the neck, ulceration of the air passages and pulmonary tissue, etc. The air enters the pulmonary tissue by means of a tear in the alveoli or bronchi. In mild cases symptoms may be in abeyance; in severe cases there may be very urgent dyspnœa and cyanosis, usually of sudden onset (diagnostic importance of the etiological factors). Examination may reveal intrathoracic creaking and crepitation (perhaps synchronous with the action of the heart), obscuration of the heart dulness and sounds and of the breath sounds, and sometimes large areas of tympany, etc.

Prognosis.—True emphysema is a disease of long duration, upward of twenty years in many cases. Complete recovery is scarcely to be hoped for, but the patient may be much benefited, his condition made bearable, and his life prolonged. Death usually results from cardiac failure or secondary infections, such as pneumonia, bronchopneumonia, tuberculosis, hemoptysis, etc.

Treatment.—The treatment virtually is that of chronic bronchitis. Efforts should be made to improve the patient's general health; he should lead a hygienic life, avoid the causative factors (blowing wind instruments, etc.) if these be detectable, and avoid also lifting heavy weights and other severe exertion, as well as cold and the taking of cold. Life in a warm, equable climate is of the greatest service. In the absence of this, respiratory exercises, which increase the force of expiration, systematic rhythmic compression of the thorax, compressed air baths, and the breathing of rarefied air are valuable. Many patients are remarkably improved by the iodides, in addition to the remedies mentioned for chronic bronchitis. In other cases alleviation of the cardiac inadequacy is followed by much benefit; as a matter of fact in moderately or well-advanced cases the cardiac factor in the symptomatology is often overlooked; small doses of strophanthus or digitalis, and other measures recommended in failing cardiac compensation, often lead to material relief. In the event of unusual cyanosis and dyspnœa venesection is called for. Attention should be paid to the diet to obviate gastro-intestinal derangements and to correct them if they develop. General tonics, such as iron, strychnine, etc., are useful from time to time.

ABSCESS OF THE LUNG.

Etiology.—Abscess of the lung most frequently follows croupous pneumonia, aspiration pneumonia, and infectious embolism. Less commonly it follows ordinary bronchopneumonia, and other diseases of the

lung, such as tuberculosis, fetid bronchitis, bronchiectasis, glanders, actinomycosis, etc. Occasionally it results from extension of suppuration from adjacent tissues and organs, such as empyema, mediastinal abscess, suppurating bronchial lymph nodes, vertebral abscess, subdiaphragmatic abscess, hepatic abscess, etc. Traumas, perforating wounds of the lung, etc., also are likely to lead to abscess. The disease is much more common in debilitated than in healthy subjects; diminution of general bodily resistance seems to be of considerable etiological importance. Staphylococci, streptococci, pneumococci, pneumobacilli, influenza bacilli, typhoid bacilli, colon bacilli, leptothrix, etc., are the bacteria commonly encountered in the abscess.

Pathology.—There may be a single abscess (following croupous pneumonia), or there may be many abscesses (following infectious embolism). The single large abscess may involve an entire lobe, or an entire lung. When multiple, the abscesses are usually small, varying in size from that of a pea to that of a walnut, wedge-shaped, and superficial; the cavity is irregular, its walls ragged, its contents purulent, and it may contain considerable necrotic lung tissue. The large single abscesses present similar appearances on a more extensive scale. When the abscess reaches to the periphery the superjacent pleura is commonly covered with a fibrinous exudation. Perforation of the abscess into a bronchus and the discharge of its contents externally is not uncommon. Perforation may also occur into the pleura (causing empyema or pyopneumothorax), or into the pericardium, etc. When old, the walls of the abscess cavity may be very thick, and bands of cicatrizing connective tissue may extend varying distances into the lung.

Symptoms.—The symptoms are often for some time obscured by those of the primary disorder—pneumonia, sepsis, etc. The small multiple abscess may remain altogether undetected. The larger abscesses, those following pneumonia, for instance, are often suggested by the non-occurrence of the crisis and an aggravation of the general symptoms, septic temperature, chills, sweats, leukocytosis, etc. But the diagnosis may not be certain until the appearance of the characteristic sputum—purulent, yellowish, greenish, or brownish (from admixture of blood), malodorous (though never as fetid as in gangrene or fetid bronchitis, and occasionally having little or no odor), and containing fragments of necrotic lung tissue, elastic fibers, fatty acids, hematoidin crystals, bacteria, etc. Sometimes a large amount of purulent sputum is expectorated at one time, whereupon the physical signs of cavity formation may become apparent—in which event the diagnosis is no longer in doubt. Often large amounts of sputum are raised early in the morning, or at other times upon the assumption of some particular posture (which favors drainage).

Diagnosis.—The diagnosis, as a rule, is easy when the symptoms and signs are fully developed—the history, the fever and other signs of sepsis, the leukocytosis, the characteristic sputum, and the physical signs of cavity formation.

Prognosis.—The prognosis is always grave. Recovery sometimes takes place when the abscess has followed pneumonia or empyema. Multiple abscesses are generally part of a pyemic process that leads to death.

Treatment.—As a rule, medicinal treatment is of minor importance, and consists of attention to the general health of the patient, the administration of stimulants, such as iron, quinine, strychnine, whiskey, etc., and the use of nutritious food. Hope of success lies in surgical measures which should be undertaken as soon as the abscess can be definitely localized.

GANGRENE OF THE LUNG.

Etiology.—Gangrene, the putrefaction of necrotic lung tissue, occurs under a variety of circumstances. An essential factor appears to be reduction in the general bodily resistance, whence it is found especially in debilitated subjects, alcoholics, diabetics, syphilitics, after severe infectious diseases (typhoid and typhus fevers, smallpox, scarlatina, etc.), in the subjects of severe mercurial and lead poisoning, etc. It follows a variety of local disorders in the lung, of which the most important are the different forms of pneumonia, especially, however, aspiration pneumonia following the entrance of foreign bodies in a bronchus. The secretion in putrid bronchitis, the contents of a tuberculous or bronchiectatic cavity, or of a lung abscess or a necrotic echinococcus cyst, etc., may induce gangrene in adjacent parts of the lung. A similar result may follow rupture into a bronchus or the lung of an empyema, a carcinoma of the œsophagus or the stomach, a gastric ulcer and subdiaphragmatic abscess, or a vertebral abscess. Infectious embolism or infection of a benign embolus, pressure of an aneurysm of the aorta, perforating wounds of the lungs and other traumas, etc., also may lead to gangrene of the lung. Staphylococci, streptococci, colon bacilli, Bacillus pyocyaneus, Micrococcus tetragenus, and the leptothrix are the organisms most commonly found in lung gangrene.

Pathology.—Two forms of gangrene may be distinguished: (1) The circumscribed, and (2) the diffuse. The circumscribed form is more common in the lower lobes, and in the right lung; but it may be bilateral; the diffuse form is most common in the right upper lobe. The diffuse form is very rare, and occurs usually after lobar pneumonia, bronchitis, or embolic obstruction of a large branch of the pulmonary artery. The gangrenous area, which may involve an entire lobe, may be indistinctly outlined, firm, and greenish black in color; more commonly it is soft, pulpy, and extremely fetid; with the progress of the process large cavities are formed, traversing which the remains of bronchi and bloodvessels may be seen. The surrounding lung tissue exhibits the changes of well-marked inflammation. The circumscribed form occurs most commonly after multiple infarction or aspiration pneumonia. The foci of gangrene

are multiple and reach often to the pleural surface of the lung; they are usually rather sharply circumscribed, irregular in shape, greenish black in color, and extremely malodorous; as the process advances, irregularly shaped cavities with shreddy walls are formed. Should these cavities communicate with a bronchus, as occasionally happens, their contents may be expectorated. The foci of gangrene are surrounded by areas of inflammation; later they may become sequestrated and encapsulated, and healing may ensue. Occasionally, rupture into the pleura occurs, with the development of pyopneumothorax. Metastatic emboli, especially to the brain, are not uncommon.

Symptoms.—The characteristic symptoms consist of fetid expectoration and fetor of the breath; but these do not develop until the putrefying lung tissue communicates with a bronchus. The symptoms, therefore, for some time are those of the primary disease. The gangrene may be inaugurated by chills, high fever, and marked prostration. Soon, however, fetor of the breath is observed, especially after coughing, and the sputum becomes characteristically fetid, and abundant, and when allowed to stand separates into three layers—an uppermost discolored, frothy layer; a middle clear, greenish or brownish, serous or mucous layer; and a lowermost layer (sediment), consisting of necrotic lung tissue, pus, fibrin, elastic tissue, fatty acids, tyrosin, bacteria, etc. Occasionally there is also blood. As a rule, the patient becomes markedly prostrated, emaciated, and septic, and soon dies of exhaustion or a severe hemorrhage.

Physical Signs.—The physical signs are often obscured by those of the primary disease. Distinctive signs consist of evidences of solidification (circumscribed foci or a large area) followed by those of excavation.

Diagnosis.—The diagnosis virtually rests upon the fetor of the breath and of the expectoration—which can scarcely be mistaken. Abscess of the lung may be excluded by an examination of the sputum. Fetid bronchitis may be distinguished by its slower course, less fetor, and absence of lung tissue in the sputum.

Prognosis.—The prognosis is grave. Death usually ensues, but recovery may take place—less likely in the diffuse than in the circumscribed form.

Treatment.—Treatment is usually limited to supporting the patient by good nutritious food and the use of stimulants—iron, quinine, whiskey, strychnine, etc. Efforts should be made to lessen the fetor by sprays of carbolic acid, or by inhalation, of creosote, carbolic acid, guaiacol, etc. If the gangrenous area can be accurately localized, recourse may be had to surgical intervention with a view to drainage, and with hope of good result.

TUMORS OF THE LUNG.

Etiology.—Tumors of the lung may be primary (rare) or secondary (more common). Their etiology is that of tumors in general. Of the

primary tumors, carcinomas developing from the bronchial mucosa (cylindrical epithelioma) or from the alveolar epithelium (squamous, medullary, or scirrhus carcinoma) are the most common; sarcoma (lymphosarcoma, round-cell, spindle-cell) and endothelioma, developing from the bronchial lymph nodes or the interstitial tissue of the lung, are less common; still rarer are fibromas, chondromas, osteomas, lipomas, lymphomas (of Hodgkin's disease), etc. Secondary tumors comprise carcinoma, sarcoma, endothelioma, and hypernephroma. The primary growths occur with greatest frequency in the breast, the digestive tract, the genito-urinary tract, and the bones. Metastasis to the lungs occurs usually by way of the bloodvessels, unless the primary growth is situated near the root of the lung, in the œsophagus, for instance, when metastasis may occur by way of the lymphatics.

Pathology.—Primary tumors generally involve one lung (the right), and the upper lobe more frequently than the lower; but they may involve both lungs. They may occur as small, firm, sharply circumscribed masses, or as large infiltrating masses. Primary carcinoma is usually a large, firm, whitish or grayish mass, often sharply circumscribed; sometimes a diffuse infiltrating mass; sometimes a series of small nodules, distributed from the hilum along the bronchi and the vessels by way of the lymphatics. Often these nodules reach the pleura, where they form flat irregular masses; marked thickening of the pleura may occur. Carcinoma shows a tendency to soften and to give metastasis (80 to 90 per cent. of the cases) to the adjacent lymph nodes, mediastinum, liver, brain, bones, etc. Secondary carcinoma occurs as small miliary or larger whitish or grayish nodules (resembling tubercles) scattered throughout both lungs. Rarely they involve only the pleura. Sarcoma presents much similar appearances. With the tumor of the lung there is often associated bronchitis, bronchiectasis (with hemorrhagic transudate), etc.

Symptoms.—The primary growth may cause no distinctive symptoms until it is far advanced. When there is extensive involvement of the lung there is usually substernal oppression, cough, dyspnœa, sometimes fever and sweats, and later emaciation. In addition there are many symptoms referable to irritation and pressure by the growth—cough (bronchitis or pressure on the bronchus); pain (involvement of the pleura); dyspnœa (pressure on the trachea); changes in the voice (pressure on the recurrent laryngeal nerve); slow pulse or rapid pulse (pressure on the pneumogastric nerve); cyanosis and œdema of the face or an arm (pressure on the veins); etc. The sputum is often quite characteristic, resembling red-currant jelly or prune juice; it may contain shreds of the pulmonary growth. Ultimately the patient becomes anemic and cachectic and dies of exhaustion. Secondary growths in the lungs are suggested by the development, in one who has or has had a primary tumor, of cough, dyspnœa, cyanosis, and blood-stained sputum; but since the primary growth is not always apparent, the diagnosis may be for a long time in abeyance.

Physical Signs.—The physical signs depend upon the size and seat of the tumor. At times there are only the signs of bronchitis; again, only moderate impairment of the percussion resonance. When the growth is extensive there may be fixation of the affected side (with compensatory activity of the other side), prominence of the affected side or distinct bulging, and widening of the interspaces. The percussion note may be normal when the foci are small and scattered; when there is a large localized mass there is dulness, which usually is very irregular in outline. Fremitus and the breath sounds may be diminished (blockage of the bronchi), or increased, that is bronchial breathing (unobstructed bronchi). The adjacent viscera (heart, liver, spleen, etc) may be displaced. The signs of pleural effusion may develop. The supraclavicular or the axillary lymph nodes may become enlarged (sometimes the first trustworthy clue to the correct diagnosis).

Diagnosis.—The diagnosis is often difficult, especially in the primary cases. In the secondary cases, attention to the history of the patient and a study of the symptomatology and physical signs often suffice for a correct diagnosis. Care should be taken to exclude bronchitis, chronic interstitial pneumonia, tuberculosis, pleurisy with effusion, etc.

Prognosis.—The prognosis is hopeless. Death usually ensues within a year or eighteen months.

Treatment.—The treatment consists in humanitarian efforts to promote the comfort of the patient and relieve his distressing symptoms.

DISEASES OF THE PLEURA.

PLEURITIS.

(*Pleurisy.*)

Pleuritis may be variously classified; it may be primary (rarely) or secondary (usually); acute or chronic in onset and course; circumscribed or diffuse; fibrinous, serofibrinous, purulent, or hemorrhagic as regards the character of the exudate; and tuberculous, streptococcic, staphylococcic, pneumococcic, etc., as regards the specific etiological factor. Inasmuch as an etiological classification is scarcely feasible at the bedside, the following clinical classification will be found serviceable: acute and chronic pleuritis; of the acute, the following forms: fibrinous, serofibrinous, purulent, and hemorrhagic; and of the chronic forms: serous and adhesive.

FIBRINOUS PLEURITIS.

(*Dry, Plastic, or Acute Adhesive Pleurisy.*)

Etiology.—Dry or fibrinous pleuritis seems to occur sometimes as an independent, or primary, disorder following exposure to cold and damp,

especially in alcoholic subjects; in most cases, however, it is a secondary process. It is most common in frank or latent pulmonary tuberculosis, but occurs also in other diseases of the lungs, such as bronchitis, pneumonia, bronchopneumonia, pulmonary abscess and gangrene, etc.; in infections of adjacent organs, such as pericarditis, endocarditis, mediastinitis, disease, including traumatism, of the bony framework of the chest, etc.; in distant infections (hemogenic infections), such as of the abdominal and pelvic organs, tonsillitis, etc.; in general infections, such as septicopyemia, rheumatism, typhoid fever, influenza, etc.; and it is a common occurrence in debilitated and asthenic states generally, such as advanced cardiac and renal disease, etc. The specific etiological factor is usually the tubercle bacillus, less commonly staphylococci, streptococci, pneumococci, etc.

Pathology.—The lesions may be more or less diffuse, but they are usually circumscribed, especially to the base of the lungs anteriorly and laterally, but often also at the apex (frequently overlooked). The inflamed area becomes hyperemic, swollen, lustreless, and finally granular in appearance; and the seat of a more or less abundant, thick or thin, usually slightly adherent fibrinous exudation. In reality the exudation is serofibrinous, that is, there is always some fluid exudation, but this is often minimal (dry pleurisy), and the excess of fibrin causes agglutination of the opposed pleural surfaces. Ultimately the exudation undergoes organization with the formation of permanent adhesions and pleural thickening.

Symptoms.—Symptoms may be almost if not entirely in abeyance, since pleural adhesions constitute a very common necropsy finding apparently unassociated with notable symptoms during life. In some cases, however, especially following exposure, or traumatism of the chest, or in the course of tuberculosis, etc., there is a sudden pain in the side (pleural stitch), perhaps slight cough (which, as well as deep inspiration, increases the pain), and slight fever. The pain may occur at the site of inflammation, or laterally or anteriorly where the movement of the lung is greatest, or it may be referred to the peripheral distribution of the involved intercostal nerves (anterior aspect of the lower thorax or the upper abdomen). Physical examination discloses restriction of the movements of the chest on the affected side and the pathognomonic friction sound. This is a jerky or interrupted, rubbing, rasping, creaking, or new-leather sound, audible most commonly during inspiration, especially at the end of inspiration, but sometimes during inspiration and expiration (to-and-fro sound); it seems to be near the auscultating ear, superficial, and it may be increased in intensity by pressure, as by the stethoscope; it often disappears temporarily after the patient has taken several deep inspirations (smoothing of the rough and irregular pleural surface?); and it is not always audible at the site of the pain (but rather laterally). Occasionally the frictions may be palpable. The breath sounds are usually feeble, from restriction of the movements of the chest.

Diagnosis.—The "stitch in the side" and the pleural frictions make the diagnosis unmistakable. In intercostal neuralgia there are points of tenderness along the course of the intercostal nerves, and an absence of fever and of pleural frictions. In pleurodynia also there is an absence of fever and of pleural frictions.

Prognosis.—Inasmuch as dry pleurisy is rarely a primary disorder, the prognosis is that of the attendant condition. The pleural manifestations, as a rule, subside spontaneously; but it is important to bear in mind that dry pleurisy may be merely the beginning of serofibrinous pleurisy.

Treatment.—In addition to attention to the primary disorder, the treatment should consist of counterirritation (iodine, blisters, or a mixture of equal parts of menthol, camphor, and chloral), opium or morphine if the pain is very severe, and other measures to be mentioned presently under serofibrinous pleuritis.

SEROFIBRINOUS PLEURITIS.

(*Pleurisy with Effusion.*)

Etiology.—There is no sharp line of demarcation between fibrinous and serofibrinous pleuritis; they represent different stages of the one process—alike etiologically (bacteriologically) and pathologically. Pleurisy with effusion, however, is most common in young male adults, apparently previously healthy; it is often believed to be a primary disorder, developing after exposure, an alcoholic debauch, etc. But these act likely merely as predisposing factors, since there is good evidence that at least 75 per cent. of so-called primary or cryptogenetic pleuritides are tuberculous in origin, the source of infection usually being calcified or fibroid bronchial lymph nodes (more or less quiescent tuberculosis). In children most cases of pleuritis follow pneumonia.

Pathology.—As a rule, the disease is unilateral, slightly more common on the right side than on the left. In the early stages the lesions are identical with those of fibrinous (dry) pleurisy. Soon, or in some cases from the beginning, a more or less abundant fluid exudate is poured out—thin, serous, alkaline, yellowish, albuminous (3 to 7 per cent.), usually at least slightly turbid, and often containing fibrinous flocculi; in some cases the fluid has a greenish tint; or it may be purulent or hemorrhagic (purulent and hemorrhagic pleuritis). The fluid varies in amount up to 3 to 5 liters; and it may be entirely free, or more or less encapsulated—in a single or a multilocular enclosure. Special localization of the lesions warrants the terms pericarditic pleuritis, mediastinal pleuritis, diaphragmatic pleuritis, interlobar pleuritis, etc. The presence of a large amount of fluid leads to distention of the affected side of the chest, and to compression and displacement of adjacent organs—the lungs, the mediastinum, the heart, the diaphragm, the liver, the spleen, etc. As a rule, the fluid is absorbed spontaneously or it is removed by thora-

centesis; following which the opposed surfaces of the pleuræ become adherent and ultimately, through organization, permanently united, leading to obliteration of the pleural sac. The new fibrous tissue contracts and gives rise to more or less distortion of the affected side of the chest, retraction of the ribs, narrowing of the intercostal spaces, scoliosis (convexity toward the healthy side), displacement of adjacent organs, especially the heart (retraction toward the affected side). In the course of time these may be almost if not quite completely overcome, especially if not marked originally; in other cases the pleural adhesions may become extraordinarily thickened and effectually prevent normal expansion of the lung, or sending prolongations into the lungs they may lead to a form of chronic interstitial pneumonitis. The other lung in these circumstances becomes the seat of so-called compensatory emphysema and the corresponding side of the chest becomes notably enlarged.

Symptoms.—The onset of the disease may be frank or insidious. In some cases the symptoms are not distinctive, scarcely even suggestive, and the disorder is disclosed only by physical examination (latent pleurisy). In other cases there is a gradual onset, with symptoms referable to the chest. In still other cases the onset is more or less abrupt, and attended by pain in the side, cough, and fever, as in acute fibrinous pleurisy; not infrequently there is a chill, or chilliness, which may last some hours or recur, and there are usually such general manifestations of infection and toxemia as headache, malaise, anorexia, etc. The pain is often extremely severe, stabbing in character, and localized to the side or the peripheral distribution of the involved intercostal nerves, although it may radiate to the abdomen, the opposite side, etc.; it is aggravated by cough, breathing, pressure, and change of posture. The cough is usually hard and dry; occasionally there is slight mucous expectoration. The breathing is usually rapid and superficial, less from dyspnœa per se than from pain set up by deep inspirations. The fever is variable, and rarely exceeds 101° to 102°; at the end of a week or ten days, as a rule, there is some evidence of lytic decrease of the temperature, but slight fever may continue for several additional weeks, and exacerbations from time to time are not uncommon. In unusual cases there may be no fever (probably, however, some fever, if sufficiently often searched for). As the serous exudate is poured out, and the inflamed and sensitive pleural surfaces are separated (the fourth to seventh day, as a rule), the pain and cough lessen and may disappear altogether; but should the amount of fluid be considerable, 200 c.c. in a child, 800 c.c. or more in an adult, symptoms due to the presence of the fluid as such and to the consequent compression and displacement of adjacent viscera develop. These comprise especially dyspnœa, cyanosis, and frequency and smallness of the pulse, which may or may not be present when the patient is quiet, but assuredly develop upon exertion.

Physical Signs.—Pleurisy may be suggested by an initial "stitch in the side" and fever, but the diagnosis, especially of effusion, is impossible in the absence of a physical examination. In the early (dry) stage the

patient is likely to lie on the healthy side to avoid increasing pain by pressure on the affected side, but when the effusion becomes large he is likely to lie on the diseased side, so as to allow free and unrestricted expansion of the unaffected side; but he may lie on his back or sit semierect in bed. When effusion has occurred the affected side is usually enlarged visibly and measurably (normally the right semicircumference is about 2 cm. greater than the left); the interspaces are on a level with the ribs or bulging. The respiratory movements of the chest and diaphragm on the affected side are lessened or abolished, the functional incapacity of the diaphragm showing well in the absence of Litten's sign and upon fluoroscopic examination. In typical cases (there are many atypical cases) directly over the fluid there are dulness on percussion, increased resistance, diminished or absent tactile and vocal fremitus, and weak or absent breath sounds; the higher the upper limit of fluid (the greater the amount, as a rule) the more marked are these signs, and they increase in intensity from above downward. The tactile and vocal fremitus (normally more marked at the right apex and often throughout the right lung) may be normal above the fluid in moderate-sized effusions; generally they are increased directly above the fluid (at the upper limit of dulness) and toward the vertebræ on account of compression of the lung; they may be well marked or increased also in the event of adhesions binding the lung to the chest wall, and apparently in children from unknown causes (perhaps small chests and considerable elastic intrapleural tension).

The upper limit of the dulness varies with the height of the fluid (which may or may not be proportionate to the amount), but it does not directly indicate the level of the fluid, since fibrinous exudate and compressed lung above the fluid also cause dulness. In small and moderate effusions the upper limit of the fluid (and of the dulness) varies, being influenced by intrapleural capillarity, retraction of the lung, and the position of the patient during the development of the fluid. Small effusions (less than 500 c.c.) may cause a small area of dulness posteriorly near the angle of the scapula, in the axilla, in Traube's semilunar space, or in the cardiohepatic (Ebstein's) angle. A significant feature of the upper limit of moderate effusions is their irregular outline; often this is low near the vertebræ, rises near the posterior or the midaxillary line, and falls again in front (the S-shaped curve of Ellis and Garland), due in part to intrapleural capillarity and the lateral-prone posture of the patient, especially during the period of development of the fluid. Had the patient been erect, the upper limit of dulness is more likely to be nearly horizontal. In very large effusions the dulness may extend up to or above the third rib in front and the spine of the scapula posteriorly. In perhaps the majority of cases (despite prevailing opinions to the contrary), the upper limit of dulness does not change with change in the posture of the patient, because the apposed surfaces of the pleuræ are usually adherent just above the fluid; in the less common cases (few limiting adhesions) postural change in the dulness may occur after a short time and after

a number of deep inspirations. There is no respiratory change in the upper limit of dulness. Above the dulness there is usually a vesiculotympanitic note (Skodaic resonance), due to relaxation o the lung; or the note may be normal. The common vesiculotympanitic note near the vertebræ, in the depression of the S-shaped curve of Ellis-Garland, may be accentuated and increased in large effusions by unusual compression of the lung. A triangular area of dulness along the vertebræ on the side opposite the effusion may be observed in moderate or large effusions (Korányi-Grocco triangle), and is due to displacement and compression of the mediastinum by the expanding pleural sac and removal of the resonant lung from the neighborhood of the vertebræ. Williams' tracheal tone and the so-called cracked-pot sound may sometimes be elicited in the first and second intercostal spaces in the event of considerable lung compression. Percussion, as well as palpation, also discloses displacement of the adjacent organs, notably the heart and the liver, less frequently the spleen and the stomach. The displacement of the heart may be extreme, but torsion does not occur, and the pulsation sometimes observed far to the right in left-sided effusions must not be interpreted as the apex beat. The lower border of the liver may reach to the umbilicus or beyond in large right-sided effusions. The lung on the unaffected side is commonly hyperresonant from compensatory inflation; it may encroach upon the normal dulness of the heart, liver, or spleen; but on the left side (in right-sided effusion) heavy percussion may bring out a tympanitic note from the stomach and intestines, especially if they be distended.

The weak or absent breath sounds usually heard over the fluid (or dull area) may be modified by attending circumstances. As a rule, in moderate or large effusions, the breath sounds decrease in intensity from above downward, and low down are totally inaudible; but at the upper level of the dulness, bronchovesicular or bronchial breathing is common from compression of the lung and non-activity of the alveoli; in very large effusions this bronchial breathing may be audible only below the clavicle and in the supraspinous fossa, or no breath sounds may be audible. In some cases breath sounds, usually more or less feeble bronchial breathing, may be heard directly over the dull area. This is due in some cases to the layer of fluid being thin (less than 2 cm.), to adhesions binding the lung here and there to the costal pleura, and to conditions favoring conduction (perhaps related to intrapleural tension). The last mentioned seem to be especially operative in children in whom the breath sounds in pleural effusion are often quite as loud and bronchial in character as they are in pneumonia; and they may be associated with bronchophony. Egophony is sometimes present, usually over the upper half or third of moderate-sized effusions. The whispered voice (pectoriloquy) is said to be transmitted (direct auscultation) through a serous, but not through a purulent effusion (Baccelli's sign). Pleural frictions are commonly audible at the upper limit of dulness; and moderately loud consonating rales are rarely audible over the dull

area in the event of the breath sounds also being audible. A systolic murmur may develop toward the base of the heart, being due to anemia, fever, or pressure on, or torsion of, the great vessels. Usually there is a slight leukocytosis—10,000 to 15,000.

Diagnosis.—The diagnosis of pleural effusion is dependent wholly upon the physical signs, of which the following are the most important: Unilateral increase in the size of the chest, immobility during respiration, diminished or absent tactile fremitus, dulness on percussion, and displacement of the adjacent viscera (heart, mediastinum, liver, etc.). Pneumonia (sometimes suggested by bronchial breathing, bronchophony, etc.) may be excluded by the absence of the characteristic mode of onset of pneumonia (initial chill, high fever, dyspnœa, and rusty expectoration), and by the more marked dulness and greater resistance, absence of tactile fremitus, and the displacement of adjacent viscera that occur in pleural effusion. Temporary absence of tactile fremitus and of bronchial breathing sometimes occur in pneumonia, being due to plugging of a large bronchus, but coughing usually dislodges the obturating plug of mucus and fibrin, and tends immediately to clear the diagnosis. In the event of doubt, and with a view to determine the nature of the pleurisy, resort may be had to exploratory puncture and the removal of a small amount of fluid. Hydrothorax may be excluded by the absence of its special etiological factors (cardiac and renal disease, advanced cachectic states, etc.), by the free mobility of the fluid (which is not confined by adhesions), and by examination of some of the fluid removed by an exploratory needle. Pulmonary and pleural tumors, which may be attended with pleural fluid, may cause diagnostic embarrassment, but they give rise to irregular dulness, the general evidences of tumors (anemia, cachexia, etc.), to obvious local growth or metastasis (mediastinum, cervical glands, etc.), and often to a hemorrhagic, sometimes chyliform fluid, in which particles of the tumor or tumor cells may be found. A pericardial effusion may be excluded by the preponderance of physical signs posteriorly rather than anteriorly, the absence of the especially significant physical signs of pericardial fluid, the presence of a displaced rather than an obscured heart, and the less-marked evidences of cardiac embarrassment (dyspnœa, cyanosis, feeble heart sounds, weak pulse, etc.).

Situation of the Exudate.—The foregoing description applies to the majority of cases, in which the fluid involves more or less of the pleural sac, and extends from below upward. In other cases the lesions are distinctly circumscribed and the fluid sacculated. This may occur anywhere, but especially anteriorly and in the axilla; it gives rise to the physical signs (already mentioned) over a restricted area Diaphragmatic pleurisy gives rise to pain and tenderness in the epigastrium (especially at the xyphoid), which may radiate to the back; often it is very severe and simulates angina pectoris. There is often distressing hiccough and spasm of the upper part of the rectus muscle; perhaps marked dyspnœa and dysphagia. The fluid accumulating

between the diaphragm and the diaphragmatic surface of the lung may not cause noteworthy dulness, but only the signs of local compression of the lung and displacement of the adjacent viscera. The fluid may be recognized by radiographic examination. Interlobar pleurisy (usually postpneumonic and suppurative) may be detected by an area of dulness corresponding to the fissure between the lobes, radiographic examination, and in most cases the symptoms of lung abscess or empyema.

Nature of the Pleuritis.—The nature of the pleurisy may be recognized by a chemical, microscopic, cytological, and bacteriological examination of the fluid. The fluid, as a rule, is pale amber in color, slightly cloudy, alkaline, has a specific gravity of 1015 to 1024, contains 3 to 7 per cent. of albumin, and it usually clots after removal. It contains usually a few erythrocytes and leukocytes, and endothelial cells (mostly fatty) in varying proportion. Cytologically preponderance of lymphocytes is indicative of the tuberculous nature of the pleuritis; preponderance of leukocytes, of pyogenic infections; and preponderance of endothelial cells, of mere transudation or malignancy. The tuberculous nature of the process may be further determined by obvious involvement of a lung or another serous membrane, by a positive response of the patient to a tuberculin test, and by inoculating lower animals with some of the fluid; later this becomes apparent in about one-third of the cases by the patient developing tuberculosis.

Prognosis.—In uncomplicated cases the immediate prognosis is good, since the general tendency of the disease, despite the common tuberculous origin, is toward recovery. The usual duration is from three to six or eight weeks. The fever, as a rule, subsides at the end of ten days or two weeks, but recurrences and exacerbations are not uncommon. With the decline of the fever the fluid tends to become absorbed (apparent in lessening of the physical signs of fluid and the development of redux crepitation). This may occur very slowly, and it may not occur at all, although resorption may occur even after months; recurrence after removal is common. Following absorption or removal of the fluid, the pleural sac, in whole or in part, becomes obliterated by fibrous adhesions; these, if thin and non-indurative, are well borne, but if thick and cicatricial they lead to induration of the lung, distortion of the chest, and displacement of the adjacent viscera, which, together with the later possibility of the development of pulmonary tuberculosis, renders the ultimate prognosis doubtful. Sudden death sometimes occurs from syncope following exertion, myocardial degeneration, mechanical embarrassment of the circulation from displacement of the heart and pressure on or torsion of the vessels, pulmonary embolism, thrombosis, or œdema; or it may follow sudden withdrawal of a large amount of fluid.

Treatment.—The patient should be confined to bed in a well-ventilated room or out-doors, and placed on a liquid or semisolid, easily digestible diet. The bowels should be thoroughly evacuated (calomel

or blue mass and a saline cathartic), and they should be kept freely open throughout the continuance of the disease. In the early stage pain usually demands relief, and it should be relieved—by an ice bag, dry cups, or strapping the side (as in fracture of the ribs); or a hypodermic injection of morphine may be given (it should not be withheld when needed); or one may use repeated small doses of powdered opium or extract of opium in a pill during the day, and, if needed, in addition administer a Dover's powder in the evening. The use of opium is sanctioned by long usage; it not only relieves the pain and the distress of breathing, but it is also believed to exercise a favorable, so-called antiphlogistic action on the capillary circulation of the pleura (and other serous membranes), and thus to control the inflammatory phenomena. Small doses of quinine as recommended in pneumonia, seem to be a useful addition. In some cases, of supposed rheumatic origin, salicyl preparations (salicylic acid and its salts, salol, salicin, aspirin, etc.) appear to do good; at all events, in sufficient dosage they tend to relieve pain. During the early fever days of the disorder the comfort of the patient may be promoted by simple febrifuge mixtures (potassium citrate, spirit of nitrous ether, solution of ammonium acetate, etc.).

When the stage of effusion has been reached one may await the spontaneous absorption of the fluid, attempt to hasten its absorption by medicinal means, or remove the fluid by thoracentesis. In general there is a tendency toward the spontaneous absorption of the fluid; but this often requires considerable time: it may not occur for months, and it may not occur at all. Attempts to hasten the absorption of the fluid are sometimes made—by counterirritation (iodine and blisters locally, and the iodides internally); by using a dry diet (very little fluid at or between the meals) and provoking copious bowel movements (concentrated saline cathartics in the morning before breakfast)—so-called Hays' method; and by the use of a chloride-poor diet. More or less satisfactory results sometimes attend these measures, but, as a rule, they are disappointing.

The most efficacious treatment consists in withdrawal of the fluid by thoracentesis. In moderate-sized and large effusions this may be done at any time with good results; but it is especially indicated as soon as the fluid appears to be not increasing in amount. This may be as early as the tenth day or thereabout, and at a time when the patient still has fever and few if any subjective pressure symptoms; but early withdrawal of the fluid is indicated because it hastens convalescence; because it relieves the compressed lung and displaced and embarrassed heart and bloodvessels, and in consequence permits of the early resumption of full functional activity; and because it obviates the danger of sudden death which occasionally occurs from high intrathoracic pressure. In the event of delaying thoracentesis, there are certain positive indications for withdrawing the fluid that should never be ignored; thus, aspiration is indicated in all cases in which there is marked displacement of the heart, and immediately upon the occurrence of embar-

rassed cardiac action (rapidity, irregularity, the development of a murmur), dyspnœa, cyanosis, or syncope; in unilateral effusion, when the dulness extends to the second interspace, or if the dulness does not extend so high, upon the development of physical signs of fluid accumulating in the other side; in bilateral effusions when both sides are half full (dulness posteriorly to the middle of the scapula); and when there is no evidence of absorption of the fluid after a week or ten days of normal temperature.

The preferable site for introducing the aspirating needle is the eighth intercostal space just external to the angle of the scapula; some physicians prefer the seventh interspace in the midaxillary line. The needle should be introduced close to the upper margin of the rib so as to avoid wounding the intercostal artery. The fluid should be withdrawn very slowly; the amount withdrawn should depend, of course, upon the amount present, but also on the reaction of the patient. Very slow withdrawal will permit the withdrawal of considerable fluid, but it is rarely necessary or desirable to remove more than 1000 to 1500 c.c.; the removal of a relatively or actually small amount is often followed by spontaneous absorption of the remainder. The operation should be discontinued when the pulmonary pleura is found to impinge against the aspirating needle inserted only a short distance, and when the patient complains of a short, hacking, paroxysmal cough (the usual signal for discontinuance), pain, or syncope. A second aspiration, rarely a third, may be required. Continuous drainage is preferable to repeated operations in recurrent cases. Barr has suggested the introduction into the pleural sac, during and after the withdrawal of the fluid, of a volume of air equal to one-half or three-fourths of that of the fluid removed, to obviate the development of a negative intrapleural pressure and to permit gradual expansion of the compressed lung; and also the introduction of one dram (4 c.c.) of adrenalin chloride (1 to 1000), to contract the bloodvessels and lessen secretion. Others have suggested the injection of gases, notably nitrogen.

Thoracentesis is occasionally attended by more or less serious untoward consequences, such as wounds of the intercostal artery, the lung, the liver, the heart, or the spleen; pulmonary œdema (albuminous expectoration), which is usually due to the too rapid withdrawal of the fluid (it should be treated with morphine and atropine hypodermicly); pneumothorax, hemothorax, subcutaneous emphysema, syncope (cerebral anemia), sudden death from pulmonary or cerebral embolism, etc.

The patient's strength must be conserved throughout the course of the disease, especially during convalescence, by the use of tonics, such as iron, quinine, strychnine, cod-liver oil, the hypophosphites, etc., good food, and fresh air. Should the lung not be speedily restored to good function, pulmonary exercises designed to inflate the lung, such as repeated deep inspirations several times daily, or the blowing of water through a narrow tube from one large flask to another, should be resorted to.

PURULENT PLEURITIS.

(*Suppurative Pleurisy; Empyema.*)

Etiology.—Purulent pleuritis is usually a secondary process caused by the pneumococcus, the streptococcus, or the staphylococcus, rarely perhaps to the tubercle bacillus, the colon bacillus, the typhoid bacillus, etc. In some cases the disorder begins as a serofibrinous pleuritis and is set up by the usual etiological (bacterial) factors of this form of pleuritis; some serofibrinous exudations, perhaps originally more opaque and cell-rich than is the rule, become still more opaque and cell-rich, that is, seropurulent, even distinctly purulent (the last mentioned usually the result of secondary infection set up perhaps by thoracentesis, or other factors). In other cases the process is purulent from the beginning or speedily becomes purulent. In children the pneumococcus is the common cause, most of the cases following pneumonia (metapneumonic form). In adults streptococci and staphylococci are the more frequent causes, but pneumococcic forms also occur. The common antecedent disorders are: Tuberculous pulmonary cavities ruptured into the pleura (the empyema in these cases being a mixed and not a pure tuberculous infection); tuberculosis, suppurative and traumatic disease of adjacent tissues—such as the mediastinum, the bony framework of the chest, the abdominal organs, etc. (lymphogenic infection); and distant localized infections (septicopyemia), or general infections (scarlatina, measles, dysentery, typhoid fever, etc.), in which secondary pyogenic infections are rather common.

Pathology.—In cases with less pus and more serum the changes in the pleura are quite like those of serofibrinous pleuritis, although more marked; the pleura is thickened and covered with a thick fibrinopurulent exudate; and there is marked polynuclear leukocytic infiltration; the fluid is more opaque and contains more cells. In cases frankly purulent the fluid usually separates into a heavy corpuscular lower layer exhibiting the ordinary characteristics of pus, and a lighter upper serous layer, greenish yellow in color. In the pneumococcic cases, especially in children, the exudate is likely to be thick and creamy and to have a sweetish disagreeable odor; in the streptococcic and staphylococcic cases the fluid usually is thinner. The fluid may become fetid (fetid or putrid pleurisy), especially in cases following stab wounds of the chest, abscess or gangrene of the lung, carcinoma of the œsophagus, appendicitis, etc.

Symptoms.—In some cases the onset and the course of the disease for some time are identical with those of serofibrinous pleuritis, of which purulent pleuritis may be only an advanced stage. In other cases following antecedent symptoms of the primary disorder, the development of the pleuritis may be abrupt, with a chill, high temperature, severe pain in the side, and general evidences of toxemia. In many cases the suppurative character of the exudate is not suspected for

some time, unless the disease develops into a frank case of septicopyemia. Long continuance of fever otherwise unexplained, however, suggests the suppuration, as do also progressive pallor, weakness, and loss of flesh; this is rendered more certain by the occurrence of paroxysms of chills (or chilliness), fever, sweats, and augmented polynuclear leukocytosis.

Physical Signs.—The physical signs in general are identical with those of serofibrinous pleuritis, that is, the physical signs no not disclose the character of the exudation. However, in purulent pleuritis the intercostal spaces are more likely to be bulging than in serofibrinous pleuritis on account of paresis (seropurulent infiltration) of the intercostal muscles; in children there may be an extraordinary enlargement of the affected side of the chest; whispered pectoriloquy is less well transmitted through a purulent than through a serous exudation (Baccelli's sign); and a localized area of œdema may develop over the fluid—which is almost if not quite pathognomonic of the purulent character of a pleural exudate. In exceptional cases the purulent exudate (very rarely also a serofibrinous exudate) transmits a cardiac systolic pulsation to the chest wall (pulsating empyema) between the second and fourth ribs anteriorly, or the third and fifth ribs in the axilla, or over a very wide area. The cause of this is not definitely known, but important factors seem to be left-sided pleural exudation (it is very rare in right-sided effusions), a large amount of fluid, paresis (relaxation, due to œdematous infiltration) of the intercostal muscles, and forcible action of the heart. Some observers believe the pulsation to be transmitted from the aorta rather than from the heart. This swelling may rupture through the costal pleura and form a pulsating mass directly beneath the skin (empyema necessitatis), and it may rupture externally—usually toward the lower part of the thorax or near the sternum; or by gravitation the empyema may reach even as low as the groin; or it may rupture into the lung and be evacuated through a bronchus, the trachea, and the mouth, or into the œsophagus, the stomach, etc.

Diagnosis.—The physical signs so essential in the diagnosis of any pleural effusion do not determine the character of the fluid; but long-continued fever, local œdema of the chest wall, and recurring chills, fever, sweats, and polynuclear leukocytosis constitute conclusive evidence of the purulent character of the fluid. In any case of doubt recourse should be had to the exploring needle, and a small amount of fluid should be removed and examined bacteriologically and cytologically. In many cases the amount of fluid (pus) is small and distinctly localized—to the base, between the lobes, etc., and being detected serves to explain ill-understood fever states (delayed convalescence from pneumonia, inexplicable attacks of fever after scarlatina, typhoid fever, etc.). It is scarcely likely that a pulsating empyema will be mistaken for an aneurysm.

Prognosis.—Purulent pleuritis not properly treated is a chronic disorder tending ultimately to death. Sometimes the cases that per-

forate spontaneously or through the lung end in recovery, the fistulous tract finally healing; or the fistula may persist with more or less discomfort to the patient and perhaps eventual amyloidosis, etc. The prognosis is better in children than in adults, especially under appropriate treatment. Marked retraction and deformity of the chest usually follow in the event of recovery.

Treatment.—Purulent pleuritis is essentially a surgical disorder, and should be treated on surgical principles; as an abscess anywhere in the body, so should an empyema be promptly evacuated upon discovery and thoroughly drained. Preliminary aspiration is not necessary, unless with a view to relieve pressure pending more thorough evacuation. The basis of successful treatment is free drainage, which sometimes may be achieved by simple free incision, especially in children; in other cases it may be necessary to resect one or more ribs. Irrigation of the cavity is sometimes resorted to in cases of putrid pleuritis, but otherwise is not necessary. Following evacuation of the pus, persistent efforts must be made to cause expansion of the compressed lung—by calisthenic exercises, pulmonary gymnastics, blowing water through a narrow tube from one bottle to another, etc. Mutilating (Estlander) operations, comprising resection of several ribs, are sometimes necessary finally to close the cavity.

HEMORRHAGIC PLEURITIS.

Etiology.—In virtually all cases of serofibrinous pleuritis the fluid exudate contains a variable number of erythrocytes. Usually these pass unnoticed unless the fluid is examined microscopically; sometimes, however, the erythrocytes are in such excessive numbers as to give a more or less well-marked hemorrhagic tint to the fluid. This occurs in certain cases of the so-called primary or idiopathic pleuritis which on careful study are found to be tuberculous, as well as the more frankly tuberculous cases with the eruption of miliary tubercles (rupture of new-formed capillaries); in primary and secondary tumors of the pleura; in the pleurisy of asthenic and cachectic states, such as chronic nephritis, cirrhosis of the liver, purpura, scurvy, leukemia, and other blood dyscrasias, etc. (in which cases it is not always a true inflammation).

Pathology.—The lesions vary with the causative factors (tuberculosis, tumors, etc.). In many cases they are quite like those of the analogous serofibrinous pleuritis—with the addition of some blood to the exudate. In cachectic states the pleura often shows hemorrhagic foci (usually petechial).

Symptoms.—The symptoms are usually those of serofibrinous pleuritis, from which hemorrhagic pleuritis is not to be distinguished except by examination of the fluid. In other cases the symptoms are those of tumor of the lung or pleura, to which the physical signs of fluid in the chest become superadded. In other cases (blood dyscrasias, asthenic states)

the phenomena are analogous to those of hydrothorax, and are disclosed usually only by a physical examination of the chest.

Diagnosis.—The diagnosis is dependent upon the physical signs of fluid in the pleura and an examination of the fluid; its hemorrhagic character can at most be merely suspected before some of the fluid is withdrawn. The condition should be distinguished from hemothorax (hemorrhage into the pleural sac), which may result from traumatism and injury to the chest wall or the thoracic organs, puncture of an intercostal vessel during aspiration, rupture of an aneurysm, ulcerative processes involving the pleuræ and pulmonary vessels, etc.; the symptoms are the general manifestations of more or less rapid bleeding and the physical signs of the more or less rapid accumulation of fluid in the pleura.

Treatment.—The treatment of hemorrhagic pleuritis, in so far as it differs from that of serofibrinous pleuritis, is dependent solely upon the associated conditions and causative factors.

CHRONIC PLEURITIS.

(Chronic Pleurisy with Effusion; Chronic Adhesive Pleurisy.)

Two types of chronic pleuritis may be distinguished—the one with, the other without, a fluid exudation.

Chronic Pleuritis with Effusion.—This may develop insidiously, or it may follow acute serofibrinous pleuritis: the etiological factors, the lesions, and the symptoms may be quite like those of the acute process. Rarely the fluid persists, little if at all changed, for months, without occasioning noteworthy symptoms; often, however, there is dyspnœa, especially on exertion. The physical signs, the diagnostic criteria, and the treatment, are quite like those of serofibrinous pleuritis.

Chronic Adhesive Pleuritis.—Etiology.—Chronic pleural adhesions are encountered under varying circumstances: (1) They may represent a primary chronic, originally fibrinous pleuritis—which perhaps may develop abruptly, but more commonly comes on insidiously; the organization of the fibrinous exudation leads to adhesion of the apposed layers of the pleura and more or less widespread obliteration of the pleura sac. These are the adhesions so commonly encountered at necropsies, often in subjects who during life presented no noteworthy evidences of their presence; originally inflammatory in nature, they are later analogous to scar tissue formation elsewhere in the body. (2) Chronic pleural adhesions are also the evidences of postserofibrinous and purulent pleuritis; they represent the process of healing, of scar formation. Following absorption of the fluid exudation, the apposed layers become adherent, and the sequential process of organization and cicatrization lead to their permanent union. As a cicatrix elsewhere, the new-formed fibrous tissue undergoes more or less contraction, and, especially in cases of empyema, may lead to extraordinary retraction and

deformity of the chest, displacement of the intrathoracic organs, and fibrous connective tissue overgrowth in the lung (chronic interstitial pneumonitis, bronchiectasis, etc.). The process is usually most marked at the base, where the two layers of the pleura usually become inseparable and indistinguishable, and represented by thick fibrous connective tissue. (3) Chronic adhesive pleuritis may be definitely tuberculous; that is, both layers may be adherent, more or less, often considerably, thickened (to 5 mm. or more), and the seat of large caseous tubercles, as well as of small miliary and fibroid tubercles; the lesions may involve one or both pleura, perhaps also the pericardium, and give rise to veritable tumor masses. (4) Chronic adhesive pleuritis may be part of a general serositis (multiple serositis, polyorrhomenitis), in which the pleuræ, pericardium, and peritoneum become involved in varying combinations; the serous membrane becomes thickened and hyaline and much resembles confectioner's sugar-icing (Zuckerguss—whence the Zuckerguss-leber, -milz, -herz, -lunge, etc., of the Germans). The pleura and the other serous sacs may be totally or partially obliterated, and if partially only, there may or may not be also some fluid.

Symptoms.—The symptoms vary. In some cases there may be a dragging pain or general aching in the side, aggravated by overexertion or inclement weather; sometimes even a sharp pain occurs. Often there is more or less dyspnœa, especially on exertion; and there may be other symptoms due to associated conditions, such as cough and expectoration in chronic bronchitis, general evidences of tuberculosis, and symptoms referable to other serous membranes.

Physical Signs.—The physical signs vary with the extent of the adhesions and the degree of contraction. In mild cases there is more or less restriction of the respiratory movement of the affected side of the chest and feeble breath sounds. In more marked cases, the affected side is definitely contracted and more or less immobile, especially at the base; the other side may be distended and the seat of exaggerated compensatory respiratory movements; the tactile and vocal fremitus and the breath sounds are lessened in intensity and may be absent; the percussion note is impaired and may be quite dull; there is no respiratory movement of the percussion resonance at the edges of the lung with the phases of respiration; and Litten's sign is absent. Auscultation sometimes reveals peculiar, dry-leathery, so-called friction sounds. In advanced cases the thorax may be extraordinarily deformed, the ribs retracted and approximated, the shoulder lowered, the vertebræ curved (the concavity toward the affected side); etc.; the heart may be uncovered by the lung, and it, as well as the liver, spleen, etc., may be more or less displaced.

Diagnosis.—The diagnosis depends upon the past history of the patient, the immobility and retraction of the chest, and the impaired resonance, feeble breath sounds, and other physical signs of a thickened pleura interposed between the lung and the chest wall.

Prognosis.—The prognosis varies with the severity and extent of the adhesions and the presence or absence of complicating conditions, such as fibrosis of the lung, bronchiectasis, deformity of the thorax, etc.

Treatment.—Attention to the general health of the patient is of prime importance. The hygienic conditions must be the best attainable, and the food must be of the best quality and generous in amount. If fluid is present in the pleura it should be removed by aspiration. Regulated exercise and pulmonary gymnastics are of marked service in favoring expansion of the lung in mild or moderately severe cases; but notable improvement can scarcely be expected when the lung is encased in a thick layer of cicatricial connective tissue and the thorax is markedly deformed. In these cases, however, if the patient lives within his reserve capacity, he may survive a long time in comparative comfort. Tonics (iron, quinine, strychnine), digestants (hydrochloric acid, tincture of nux vomica, etc.), and alteratives (syrup of iron iodide, cod-liver oil, mercury, phosphorus, etc.) are often useful adjuvants.

HYDROTHORAX.

(*Pleural Dropsy; Pleural Œdema.*)

Etiology.—Hydrothorax, a transudation of non-inflammatory serous fluid into the pleural sac, is a secondary process that occurs in conditions that lead to œdema generally: disease of the heart and of the lungs (with consecutive cardiac failure) in consequence of passive congestion; and in disease of the kidneys and anemic and cachectic conditions (carcinoma, sarcoma, pernicious anemia, leukemia, scurvy, etc.), in consequence of hydremia, and perhaps disease of the capillary vessels and toxemia.

Pathology.—In cardiac cases, the process in the beginning at least is likely to be unilateral, commonly right-sided, because in addition to the general venous engorgement, local obstruction of the azygos veins or the right pulmonary veins (page 457) is added, the consequence of pressure by a dilated right auricle or vena cava; and when it becomes bilateral it is still usually most marked on the right side. In renal and anemic and cachectic cases it is usually bilateral. The fluid varies in amount from a few cubic centimeters to several liters. It is usually clear, pale amber in color, has a specific gravity of 1006 to 1015, and contains from 1 to 3 per cent. of albumin; microscopically, it contains a few endothelial cells, erythrocytes, and leukocytes. The pleura often exhibits no conspicuous changes; but in long-standing cases a low grade inflammation may be set up and materially alter the appearance of the pleura and the character of the fluid.

Symptoms.—The symptoms are those of the primary condition, to which are added the effects of pressure on the intrathoracic organs, dyspnœa (orthopnœa), cyanosis, cough (often paroxysmal and asthmatic), and marked embarrasment and enfeeblement of the circulation.

The physical signs are those of fluid in the chest, and are quite like those of serofibrinous pleuritis. The fluid, however, being thinner and not circumscribed by adhesions, readily shifts with change in the posture of the patient (pathognomonic sign of fluid).

Diagnosis.—The diagnosis depends upon the etiological factor, the physical signs of fluid in the pleura, and an examination of some of the fluid removed. It is especially important to remember that often much of the distress of failing cardiac compensation is due to a right-sided hydrothorax that is often entirely unrecognized.

Prognosis.—The prognosis is that of the primary disorder.

Treatment.—The treatment is that of the primary disorder. In many cases of failing cardiac compensation, the greatest improvement follows removal of the fluid by aspiration. The condition should always be borne in mind and searched after, and when detected, if the patient does not speedily improve, the thoracentesis should be promptly performed; delay may precipitate a fatal issue, which otherwise might be avoided.

PNEUMOTHORAX.

(*Pyopneumothorax; Serofibrinopneumothorax; Hemopneumothorax.*)

Pneumothorax, the presence of air or gas in the pleural cavity, is an exceedingly rare disorder. The causative factors are such that an inflammation of the pleura is also induced, and inasmuch as this is usually purulent, the condition in the great majority of cases is pyopneumothorax; rarely the inflammatory phenomena are analogous to those of serofibrinous pleuritis—serofibrinopneumothorax; and still more rarely the exudation is hemorrhagic—hemopneumothorax.

Etiology.—Pneumothorax (pyopneumothorax and its other modifications) is most common in males, in adult life, and on the right side. It results from the access of air (which is usually infected) to the pleura. This may occur: (1) In perforating wounds of the chest, such as traumatisms (when it may be associated with subcutaneous emphysema), thoracentesis, etc. (2) In rupture of the lung, which is the most common cause, especially the rupture of tuberculous pulmonary cavities; less commonly the rupture of a pulmonary abscess and gangrene, bronchiectatic, bronchopneumonic, and embolic cavities, echinococcus cyst, emphysematous bulla, etc.; or the rupture into the lung of an empyema, or traumatic rupture of the lung due to a fractured rib without laceration of the overlying skin; rarely perhaps violent muscular effort or coughing may cause rupture of a lung bound down by adhesions. (3) In ulcerative processes in the œsophagus, stomach, intestines, etc. (with perhaps a primary subphrenic abscess). (4) Rarely pneumothorax may arise spontaneously, in consequence of infection with Bacillus aërogenes capsulatus (Welch) or other gas-producing microorganism.

Pathology.—The process is usually unilateral; rarely bilateral. The contained air may or may not be malodorous; it varies in amount up to 2000 c.c. or more; and it is commonly associated with more or less seropurulent or distinctly purulent fluid and lesions similar to those of purulent pleuritis—all of which, soon overcoming the negative intrapleural pressure of 4 to 6 mm. of mercury, causes proportionate compression and displacement of the intrathoracic and upper abdominal organs. In rupture of a previously healthy or an emphysematous lung there may be no fluid; and in traumatic cases, as well as in cases due to ulcerative processes, there may be also blood—hemopneumothorax (or hemopyopneumothorax). The air or gas may be circumscribed or diffuse (circumscribed or diffuse pneumothorax). The point of rupture in the lung may be small or large; usually, but not always it may be found; it may close after permitting the access of air to the pleura.

The following varieties of pneumothorax may be distinguished: (1) Open pneumothorax, in which the air enters and leaves the pleura during the phases of respiration; (2) closed pneumothorax, in which the point of rupture (usually in the lung) becomes closed, and shuts off from the outside air that contained in the pleural sac; and (3) valvular pneumothorax, which permits usually the entrance, but not the exit of air from the pleura; the entrance of air usually ceases when the intrapleural pressure is sufficient to close the opening or to overcome the pressure of the entering air.

Symptoms.—Rarely, the onset is insidious, and not attended by noteworthy symptoms; the condition may be found accidentally by physical examination during life or at the necropsy. In the great majority of cases, however, the onset is sudden and characterized by severe pain in the chest, the development of (or increase of previously existing) dyspnœa and cyanosis, and not infrequently collapse, with rapid, feeble pulse, sweating, etc.

Physical Signs.—The patient is likely to lie on the affected side, to permit free and unrestrained movement of the healthy lung. The affected side is distended, and almost if not quite immobile; the intercostal spaces bulge and may transmit a downy or crepitant sensation on palpation. The tactile fremitus is markedly diminished or absent. The note obtained by percussion varies, depending upon the intrapleural tension, the tension of the chest wall (due largely to the condition of the intercostal muscles), and whether the pneumothorax is of the open or closed variety. As a rule, the percussion note is full, clear, and hyperresonant or tympanitic; it is said to have an amphoric or metallic quality in open pneumothorax (not a conclusive or trustworthy sign to be thus interpreted). Of special importance is the fact that the hyperresonant or tympanitic note extends far beyond the normal limits of the lung. Sometimes the percussion note is quite dull (said to be cases of marked intrapleural tension and contracted intercostal muscles). Often there is dulness at the base of the chest (due to concomitant fluid exudation), which shifts readily with change in the posture of the patient; but

fluid may be present without dulness, the tympany obscuring or replacing the dulness. The so-called cracked-pot sound may sometimes be elicited over an open pneumothorax. The breath sounds, as a rule, are much diminished or absent, especially at the lower part of the chest; or feeble breath sounds of a bronchial, metallic, or amphoric quality may be audible; or this quality may be well brought out by a cough. On auscultating posteriorly while an assistant with a coin taps another coin placed on the front of the chest, one may hear the so-called coin sound (bruit d'airain of Trousseau). The vocal fremitus (bronchophony) also has a metallic quality, although it is usually less intense than normally. Bronchial and other rales may be heard, and usually have a metallic quality. The metallic tinkle of Laënnec, or falling-drop sound, may be elicited by coughing, deep inspiration, or change of posture; usually attributed to the falling of a drop of fluid or fibrin on the accumulated fluid, it is not unlikely due to rales produced in or about a cavity in the lung. A peculiar whistling sound is sometimes audible and is attributable to entrance or exit of air through the pulmonary opening into the pleura; this takes on a quite characteristic quality when the opening is below the upper level of the fluid. A peculiar splashing sound (Hippocratic succussion splash) may be heard when the patient is shaken, and is pathognomonic of the presence of air and fluid; it may be heard only when the auscultator's ear is applied to the chest, but sometimes at a distance; and it may be quite apparent to the patient. In addition to the foregoing, the physical signs of displacement of the adjacent viscera, especially the heart, are present; the heart sounds, although muffled, may have a metallic quality.

Diagnosis.—The diagnosis, as a rule, is easy: the etiological factor, the mode of onset, the enlarged and immobile chest, the hyperresonant or tympanitic percussion note, the markedly diminished or absent vocal fremitus and breath sounds, the metallic quality to breath sounds or rales that may be audible, the coin sound, succussion splash, and displacement of the adjacent viscera are unmistakable. It is not likely that a careful observer will mistake the condition for emphysema. In the unusual cases in which the percussion note is dull, effusion alone may be suspected, but absence of the characteristic increased resistance to the percussing finger and of the other signs of pleural effusion should exclude error; but one must remember that fluid is usually associated with the air. Pulmonary cavity may be excluded by the absence of retraction of the chest and of sinking in of the intercostal spaces, and by the presence of dislocation of the heart, of the coin sound, of succussion splash, etc. Diaphragmatic hernia and subphrenic pyopneumothorax also must sometimes be considered in the differential diagnosis. Röntgen-ray examination often affords material help in the diagnosis.

Prognosis.—The outlook varies with the associated conditions, but it is always serious. Death occurs in about three-fourths of the cases;

within a short time (hours or days) in a few cases, within a few weeks in most of the cases (tuberculous cases). Occasionally arrest of the tuberculous process in the lung follows the development of the pneumothorax. Some cases become chronic and last for months or years. Recovery may ensue when the condition develops in a previously healthy subject.

Treatment.—In some traumatic, tuberculous, and other cases the air becomes absorbed spontaneously; the patient, therefore, should be watched, kept quiet, and rendered comfortable by the use of bromides, morphine, and other sedatives and narcotics, if necessary; if no noteworthy ill consequences ensue the pneumothorax should not be molested. Removal of the air by aspiration may lead to reopening of a healing point of rupture in the lung and convert a closed into an open pneumothorax, and induce or increase infection of the pleura. The cases of frank pyopneumothorax, as a rule, should be treated as are cases of purulent pleuritis, by incision and drainage; but one often has to take into consideration the general condition of the patient, the likelihood of the pleural fistula opening and of the disease of the lung, perhaps quiescent, becoming reactivated, the condition of the other lung, etc. The likelihood of benefit from incision and drainage are often almost if not quite counterbalanced by the possible ill consequences; in general, however, it is always wise to evacuate pus. In acute cases with marked cyanosis, orthopnœa, and feeble heart action the pleura should be incised or aspirated without delay, and morphine and stimulants administered. Otherwise the treatment is that of the associated conditions, chiefly tuberculosis.

CHYLOTHORAX.

Chylothorax, the presence of a milky, opaque, and opalescent fluid in the pleura, may be of two types: (1) True chylothorax, in which the fluid is chyle. This occurs rarely in the event of obstruction or rupture of the thoracic duct, and still more rarely in obstruction of the subclavian vein (thrombosis, or pressure from without by tumors, etc.). (2) False chylothorax (pseudochylothorax), in which the fluid is chyliform (adipose), the consequence of fatty degeneration of contained cells and of fatty (and perhaps albuminous) particles held in suspension; it occurs rarely in tuberculous, endotheliomatous, and other forms of pleuritis. Both forms of chylothorax may be associated with similar disease of the peritoneum. The *symptoms* and *physical signs* are like those of hydrothorax, serofibrinous pleurisy, or tumor of the pleura. The *diagnosis* is usually not made until the fluid has been obtained by thoracentesis. It is often difficult to distinguish chylous from chyliform fluid: chylous fluid contains sugar, which, however, has been found also in some cases of chyliform fluid; chylous fluid, as a rule, contains a small amount of fat (1 per cent, or less), while chyliform

fluid may contain 5 to 6 per cent. or more, and its fatty and albuminous granules are likely to be coarser than those of chylous fluid.

TUMORS OF THE PLEURA.

Tumors of the pleura may be primary or secondary. The secondary growths usually follow primary tumors of the lungs, or mediastinum (extension by contiguity or lymphatic distribution), or of the kidneys (hypernephroma) or other organs from which metastasis may occur by way of the bloodvessels. The primary tumors may be benign (lipoma, fibroma, chondroma) or malignant—the more common endothelioma and the rarer sarcoma (round- or spindle-cell). They occur as flat, circumscribed or diffuse, more or less soft growths; and cause moderate or considerable thickening of the pleura. Metastasis may occur to the lungs and other adjacent organs; and the growth may become visible beneath the skin. The *symptoms* are those of chronic pleurisy, with perhaps considerable local pain or distress, and progressive anemia and cachexia. The chest may be irregularly distended and retracted, and the dulness may vary in intensity here and there and be quite irregular in outline. The fluid removed by exploratory puncture is likely to be hemorrhagic, and may contain isolated tumor cells or particles of the growth. The *prognosis* is bad, the course of the disease being, as a rule, a few months only. The *treatment* is purely symptomatic—promotion of the comfort of the patient, relief of pain and distress by morphine if necessary, the removal of large collections of fluid, etc.

DISEASES OF THE MEDIASTINUM.

MEDIASTINITIS.

Mediastinitis may be acute or chronic. The chronic process is usually part of a chronic obliterative pericarditis (mediastinopericarditis), associated or not with chronic obliterative pleuritis; the symptoms are discussed in connection with chronic adhesive pericarditis.

Acute mediastinitis is sometimes divided, according to its location, into anterior and posterior, the dividing line being a plane laterally through the middle of the trachea. The process may be simple or suppurative (often two stages of the one process). It may be due to trauma; to inflammation of adjacent tissues (pleura, pericardium, lungs, œsophagus, ribs, sternum, vertebræ, connective tissue of the neck, etc.); or to infectious, especially septicopyemic, processes elsewhere in the body (hemogenic infection). The lesions are those of inflammation, often associated with considerable œdema, which may proceed to abscess formation. Either the connective tissues or the lymph nodes, or both, may be involved, and the lesions of one or the other may predominate. The

lymph nodes are especially involved in tuberculosis, syphilis, and other infections, such as pertussis.

Symptoms.—The symptoms consist: (1) Fever, and perhaps initial chill or chilliness; (2) evidences of local inflammation, such as substernal pain and tenderness, and perhaps œdema about the sternum, in the intercostal spaces, and the episternal notch; and (3) pressure effects, such as dyspnœa, dysphagia, etc. (more fully mentioned under mediastinal tumors). In the event of suppuration, irregular chills, fever, sweats, and leukocytosis may supervene; the pain may become more severe and throbbing in character, and the local effects of pressure more aggravated. The abscess may penetrate and ultimately perforate through an intercostal space, the trachea, a bronchus, the pleura, the œsophagus, etc., or it may burrow into the abdomen.

Diagnosis.—The bulging sometimes caused in an intercostal space may be mistaken for an aneurysm, especially if it pulsates and is fluctuating; but the absence of expansile pulsation, of diastolic shock, and of murmurs, and the presence of the general and local evidences of suppuration should obviate diagnostic error.

Treatment.—The treatment is surgical, and consists of evacuation and drainage.

TUMORS OF THE MEDIASTINUM.

Tumors of the mediastinum may be primary or secondary. The secondary growths (carcinomas and sarcomas) develop from extension of tumors of adjacent organs or represent metastases from more distant parts. The primary tumors comprise the exceedingly rare benign growths (fibroma, lipoma, chondroma, etc.), and the more common malignant growths, of which the most common is sarcoma; carcinoma is rare (despite older views to the contrary; in days gone by carcinoma was not well differentiated from sarcoma); rare endotheliomas have been reported, as well as simple and dermoid cysts. Sarcoma may develop from the mediastinal connective tissues or lymph nodes, or from the thymus; when advanced it is often difficult to determine the point of origin. The common types are the lymphosarcoma and the spindle-cell sarcoma; they form tumors of variable size and consistency, that more or less completely fill the mediastinum, and compress the regional structures and organs.

Symptoms.—As a rule, the symptoms develop insidiously and progress gradually. The initial symptoms in most cases consist of a hard, dry cough or gradually increasing dyspnœa, especially on exertion, and progressive failure of the general health; rarely these develop rather suddenly and soon become marked. There may be also a sense of fulness or constriction of the chest; or even actual substernal pain.

The symptoms are due almost entirely to pressure, and they vary much in individual cases. Dyspnœa may result from compression of

the trachea, bronchus, lung, or pneumogastric nerve. Spasm of the glottis, paralysis of the vocal cord, aphonia, and paroxysmal dyspnœa may follow irritation or paralysis of the recurrent laryngeal nerve (usually the left, sometimes both). The cardiac activity may be accelerated or weakened, and attacks of asthma may occur, from involvement of the pneumogastric nerve or compression of the heart. Cyanosis, œdema, and venous ectasia of the head, neck, arm, and upper chest may result from compression of the ascending vena cava or one of its main branches, and superficial anastomosis may form with tributaries of the inferior vena cava. Hydrothorax may result from local venous obstruction. Dysphagia may follow pressure on the œsophagus. Inequality of the pupils and unilateral sweating of the face may result from involvement of the sympathetic nerves. The growth occasions an irregular area of dulness beneath and beyond the sternum, or in the back. The breath sounds in a lobe or in an entire lung may be weakened from compression of a bronchus, or they may be bronchovesicular or bronchial in character from compression of the lung. The heart becomes more or less displaced. The growth may erode and finally penetrate the sternum or project through an intercostal space, in the episternal notch, etc.; and it may exhibit a pulsation (non-expansile) transmitted from the heart or the aorta; and it may give rise to metastatic enlargement of the cervical lymph nodes, etc.

Diagnosis.—The disease should be suspected in all cases of ill-understood cough and dyspnœa, especially if associated with progressive deterioration of health. Radiographic examination may prove of the greatest service, revealing a non-expansile tumor mass. The greatest difficulty is experienced in differentiating aneurysm of the aorta (page 501).

Prognosis.—Death usually ensues within six or eight months of the onset of symptoms.

Treatment.—The treatment, as a rule, is symptomatic. Attempts to remove the growth have been made and with success in tumors of the thymus, and dermoid cysts.

SECTION VIII.

DISEASES OF THE DIGESTIVE SYSTEM.

DISEASES OF THE MOUTH.

STOMATITIS.

Although different varieties of stomatitis are described, they are much related etiologically, and in many cases they represent different stages of the same anatomical process. Thus, a catarrhal stomatitis frequently proceeds to the formation of vesicles and superficial erosion (aphthous stomatitis), and this in turn to a well-defined ulceration (ulcerative stomatitis). Thrush, to which (although incorrectly) the term parasitic or mycotic stomatitis is sometimes restricted, is a catarrhal stomatitis associated with a growth of Saccharomyces (Oïdium) albicans. Gangrenous stomatitis, however, although in some respects allied to the milder forms of stomatitis, is probably a more or less independent affection.

Etiology.—Stomatitis may occur at any age, but is more frequent in poorly nourished and marantic children. Its development is much favored by insanitary surroundings, inattention to the hygiene of the mouth, mouth-breathing in patients affected with adenoids, and general debility from any cause. It is frequently associated with and follows inflammation of adjacent structures, such as the nose, pharynx, and especially the gastro-intestinal tract. The exciting causes are either infectious, toxic, mechanical, or thermic.

Infectious stomatitis may arise as a primary or a secondary process. As a secondary process it is common in certain of the infectious diseases, such as measles, scarlatina, influenza, typhoid fever, smallpox, syphilis, etc.; it occurs also in the debility of old age, as well as in the adynamia of carcinoma, tuberculosis, profound anemia, etc. A specific bacterial cause has not been isolated, but it is probable that many of the more common bacteria which have been found in association with the lesions, such as pneumococci, staphylococci, streptococci, the typhoid bacillus, influenza bacillus, the causative factors of measles and of scarlatina, certain leptothrix, etc., are answerable in different cases. A special variety of stomatitis, thrush, to which, although incorrectly, the term

parasitic or mycotic stomatis is sometimes restricted, is associated with infection of the mouth by Saccharomyces (Oïdium) albicans, an organism which, because of its polymorphism, has been described under a variety of names. Infection with this organism is usually preceded by catarrhal stomatitis, so that the disease is probably the consequence of mixed infection. It occurs most commonly in infants, but also, especially as a terminal infection, in old age, tuberculosis, diabetes, carcinoma, etc. Its development is facilitated by mouth breathing and the restricted movements of the tongue, so common in adynamic patients, which favor the lodgement and growth of the fungus. Infection may be transmitted directly, as by infected nipples, but it is usually acquired by inhalation. Further evidence of the infectious nature of some cases of stomatitis is found in the fact that a form of the disease, usually ulcerative in character, sometimes prevails epidemically in insanitary jails, tenements, asylums, barracks, camps, etc. Rarely foot-and-mouth disease, aphthæ epizoöticæ, affects the buccal mucous membrane of human beings. Some observers have maintained that the causative factor of ulcerative stomatitis in human beings is allied to that causing foot-and-mouth disease in cattle—infection being transmitted with the milk; others, such as Payne, have suggested that the organism is allied to that of impetigo contagiosa.

Toxic stomatitis results from the ingestion of irritant and corrosive poisons, such as strong acids and alkalies; from the excessive and prolonged use of alcohol, tobacco, strong condiments, etc.; and sometimes from the medicinal or other use of mercury, arsenic, lead, the iodides, the bromides, etc.

Mechanical stomatitis sometimes attends the irruption of the teeth in infants and children; it frequently results from the irritation caused by sharp foreign bodies, broken and carious teeth, the too vigorous cleansing of the teeth and mouth, etc. It is frequently associated with the consequences of inattention to the teeth and the resulting accumulation of filth, decomposing food, bacteria, incrustations, etc.—whence it is partly infectious in nature.

Thermic stomatitis results from the ingestion of overheated food and drink, as well as from burns due to other causes.

Pathology.—In mild cases of stomatitis the lesions are those of catarrhal inflammation in general—swelling, hyperemia, increase of secretion, and later some desquamation of the superficial epithelium. In some cases the inflammation may extend to the submucous tissues and give rise to thickening, œdema, and induration. In more severe cases, in addition to the hyperemia, there are localized areas of round-cell infiltration which lead to the formation of papules and later vesicles; these, losing their epithelial covering, produce circumscribed whitish patches with slightly elevated edges, from which within a short time well-defined erosions—aphthæ—develop. In more severe cases the lesions proceed to ulceration—superficial necrosis with round-cell infiltration. In thrush the mucous membrane is the seat of catarrhal inflammation, upon which, in conse-

quence of the development of Saccharomyces (Oïdium) albicans, small whitish spots resembling curdled milk and about the size of a pin's head develop. The fungus develops just beneath the uppermost layers of the surface epithelium, having first penetrated the cement substance of the most superficial cells, which in consequence of the growth become the seat of pressure atrophy. The fungus rarely penetrates to the deeper layers, but it has been known to reach the lymphatics and even the bloodvessels, and thus gain access to distant parts of the body, such as the brain, etc. The whitish patches, consisting of the fungus, desquamated and degenerated epithelium, and debris, if removed early may leave a somewhat raw surface, but having existed for several days, they may be removed with ease and leave an almost if not quite intact mucous membrane. Of discrete growth at first, the fungus later may spread to all parts of the oral and nasal cavities, and even to the pharynx, larynx, œsophagus, and gastro-intestinal tract.

Symptoms.—Clinically, the following varieties of stomatitis may be distinguished: (1) Catarrhal; (2) aphthous; (3) ulcerative; (4) membranous; and (5) thrush.

Catarrhal stomatitis (simple stomatitis, erythematous stomatitis, pultaceous stomatitis) is characterized by local pain (burning sensations), heat, redness, and swelling, especially of the lips, gums, and tongue. The tongue exhibits indentations caused by the teeth, and its swollen papillæ may be visible through a thick grayish or yellowish coating. The swollen oral mucous membrane often presents distended mucous follicles. The oral secretions, at first lessened, soon become much increased and are often irritating and acid, occasioning, especially in children, erythematous areas about the angles of the mouth. Together with decomposing remnants of food, desquamated and degenerating epithelium, leptothrix, and other bacteria, which collect about the teeth as well as on the dorsum of the tongue, they give to the breath a fetid odor. There are unpleasant sensations of taste, thirst for cool drinks is marked, and suckling and mastication are painful. General symptoms are rare, except perhaps in young children, in whom restlessness and slight fever may occur. Usually the disease runs its course in from five to seven days. Occasionally, however, especially in alcoholic subjects and inordinate smokers, the lesions tend to become subacute or chronic. In these cases, the mucous membrane of the mouth becomes dark brownish red in color, somewhat thickened and opaque in scattered areas, and sometimes fissures develop. The subjective symptoms are usually slight.

Aphthous stomatitis (follicular stomatitis, vesicular stomatitis, herpetic stomatitis) is characterized by the development of small superficial erosions or ulcerations (2 to 4 mm. in diameter) surrounded by a reddish inflammatory zone. The symptoms are those of catarrhal stomatitis, which is usually also present, but the local pain is perhaps more severe. Aphthæ may come out singly or in a group; usually all appear within twenty-four hours. They are most numerous on the tip and edges

of the tongue, and the inner aspects of the lips and cheeks; they are rare on the pharynx. They appear as small vesicles, which, usually within twenty-four hours, rupture, leaving an erosion with a grayish base and a slightly elevated edge. Within twenty-four or thirty-six hours this becomes quite sharply defined and surrounded by a narrow but distinct inflammatory zone. At this time they are quite painful, and although suggesting, they differ from, ordinary ulceration in the absence of suppuration. Rarely from confluence a large ulcer is formed. The general symptoms consist of restlessness and slight fever or merely an aggravation of the symptoms of the primary disorder—usually gastro-intestinal catarrh. The disease commonly runs its course in less than a week, although occasionally it is protracted by successive crops of aphthæ, or by extension of the lesions to the fauces and pharynx.

Ulcerative stomatitis (fetid stomatitis, putrid sore mouth, phlegmonous stomatitis, phagadenic stomatitis, stomacace) is a form of stomatitis characterized by the development of ulceration. The cases vary much in severity, from the mildest to those with extensive and widespread destruction of tissue, implicating sometimes the bone. The mildest cases may begin as a catarrhal or herpetic stomatitis, proceed to ulceration, and soon result in recovery. A single small, so-called simple ulcer, is by no means uncommon on the mucous membrane of the lips and cheeks, especially in gastro-intestinal derangements, and also it is said in nursing women. More severe lesions sometimes occur in the course of certain infective diseases, such as measles, scarlatina, typhoid fever, pertussis, etc., especially in debilitated and emaciated children; they are common in scorbutus; they sometimes appear during the course of, and sometimes as the initial manifestation of, leukemia; and they not infrequently follow the administration of metallic poisons, such as mercury, lead, arsenic, phosphorus, as well as iodine, bromine, etc. Certain severe cases often appear epidemically, especially in insanitary jails, tenements, asylums, barracks, camps, etc. The development of the affection is much favored by poor and insufficient food, inattention to the hygiene of the mouth, the presence of carious teeth, and the winter season. A specific cause seems likely, but it has not yet been isolated, although it has been said to be allied to the cause of foot-and-mouth disease of cattle and to the cause of impetigo contagiosa.

The disease begins at the margin of the gums, usually near the molar teeth, but especially about a carious tooth. The gums become red, swollen, œdematous and painful; they bleed readily, and frequently retract, thus exposing the teeth to the point of insertion in the jaw. The lesions spread to the adjacent cheek and sometimes the lip, and the mucous membrane becoming necrotic leads to the formation of a more or less extensive sphacelus and ulceration. In severe cases mucopurulent matter collects beneath the gums and gives rise to a very extensive sloughing, the exposed teeth loosen and may drop out, and inflammation and necrosis of the bone may result. In addition to the ulceration there is catarrhal and aphthous inflammation of the oral mucous membrane,

profuse salivation, extremely foul breath, enlargement of the regional lymph nodes, gastro-intestinal derangements, moderate fever (102° F.), etc. In some cases evidence of sepsis, including an erythematous (measly) rash, leukocytosis, sweats, etc., develop, and the patient emaciates rapidly. The majority of cases run their course in from a week to ten days, and terminate in recovery. Certain severe cases, however, result fatally.

In *mercurial stomatitis* (ptyalism), in addition to the inflammation of the mucous membrane of the mouth, there is also inflammation of the salivary glands. The disease begins with a metallic taste and soreness of the teeth on closing them vigorously. These are soon followed by increased flow of saliva and tenderness and sponginess of the gums. The tongue becomes swollen and flabby, the breath foul, the regional glands enlarged, and mastication and deglutition difficult and painful. Should the mercury not be stopped, well-developed ulceration and necrosis, with loosening and eventual falling of the teeth and necrosis of the bone, may ensue.

Various other forms of ulcerative stomatitis have been described. Thus, *Riga's disease* (Riga's aphtha), common in Southern Italy and sometimes occurring epidemically in young (especially marantic) children about the time of irruption of the first teeth (lower incisors), consists of the development of a grayish induration, proceeding to ulceration, situated at the frenum of the tongue. A similarly situated ulceration sometimes occurs in pertussis from the forcible projection of the tongue against the lower incisor teeth during the paroxysms of coughing. *Bednar's aphthæ* (aphthæ of the palate) which sometimes occur in young marantic, especially bottle-fed babies, consist of grayish patches of superficial necrosis proceeding to ulceration of both halves of the posterior part of the hard palate near the alveolar processes. It is usually looked upon as a pressure necrosis with subsequent bacterial infection, occasioned by the nipple or the nurse's finger. Jacobi has described a *chronic neurotic stomatis*, in some respects akin to pemphigus vegetans, occurring in neurotic individuals with hereditary predispositions, and characterized by recurring attacks of herpetic, sometimes pemphigoid, eruption. Parrot has described a form of ulcerative stomatitis occurring in debilitated, newly born children, and consisting of small ulcers situated symmetrically on both sides of the hard palate, frequently chronic, and tending to increase in size and involve the bone. Lemaistre has described, under the term *la perleche*, a condition consisting of painful fissures and elevations at the angles of the mouth, occurring epidemically, due to a micrococcus, and running its course in two or three weeks.

Membranous stomatitis (croupous stomatitis) is characterized by the formation of a false membrane. It is usually diphtheritic in character and due to the Klebs-Loeffler bacillus; but it sometimes results from the action of streptococci, staphylococci, gonococci, or Treponema pallidum. The local and constitutional symptoms are usually those of diphtheria.

Thrush (parasitic stomatitis, mycotic stomatitis, soor, muguet) is a form of stomatitis due to Saccharomyces (Oïdium) albicans, and characterized by the development of small whitish spots which tend to coalesce and spread more or less extensively throughout the oral cavity.

The disease begins usually on the dorsum of the tongue, the saccharomyces, consisting of branching filaments, developing in the superficial layers of the mucosa, and giving rise to small whitish spots that are slightly elevated. As the spots become old, they tend to become discolored, yellowish, and brownish. The lesions of catarrhal and of aphthous stomatitis are usually associated, but the mouth is dry and the oral secretions usually lessened. The general symptoms are inconspicuous, and scarcely differ from those of catarrhal or of aphthous stomatitis, although perhaps gastro-intestinal derangements, especially diarrhœa, are more common. The disease, however, occurring in the course of other diseases, is frequently overlooked on account of the absence of local symptoms. Under appropriate treatment it is usually curable within two weeks, but it sometimes runs a protracted course.

Diagnosis.—The diagnosis of the different varieties of stomatitis is usually quite obvious from an inspection of the mouth. Thrush should not be mistaken for milk curds nor for aphthous or ulcerative stomatitis, from which it is distinguished by the absence of pain and erosion or ulceration, and the presence of dryness of the mouth and the specific fungus.

Prognosis.—The prognosis is good, at least as regards the lesions in the mouth. The diseases with which the stomatitis is frequently associated, however, often terminate fatally, the stomatitis being a local terminal infection.

Treatment.—Prophylaxis is much more important than treatment as such. The hygiene of the mouth must be carefully attended to, especially in young nursing children and in the debilitated of all ages. The mouth should be carefully and repeatedly cleansed with bland antiseptic solutions, such as a saturated solution of boric acid in rose water, to which 5 to 10 per cent. of glycerin may be added. Should the disease be developed strict cleanliness is imperative. The causative factors in each case must be corrected. In the milder cases it suffices to use boric acid, borax, sodium bicarbonate, or potassium chlorate (2 per cent. solutions) to which 5 per cent. of tincture of myrrh may be added. Or one may use a mouth wash of potassium permanganate (1 per cent.) or of hydrogen peroxide (2 to 10 per cent.); or the following:

℞—Thymol	5 minims	0	3
Oil of eucalyptus	10 minims	0	6
Benzoic acid	1 dram	4	0
Distilled water	3 ounces	100	0

—Miller.

Cool drinks are usually accepted gratefully. Should aphthæ develop they should be touched with the solid stick of silver nitrate (one application of which usually suffices to effect a cure), or a 5 per cent. solution of

silver nitrate may be used. Potassium chlorate is looked upon as almost a specific in aphthous and ulcerative stomatitis. Locally it may be used in powder form, or in a 2 to 5 per cent. solution as a mouth wash; internally, 1 to 3 grains (0.06 to 0.3 gram) carefully to a child, 10 to 20 grains (0.65 to 1.3 gram) to an adult, may be given three times a day. The following formula is serviceable:

℞—Potassium chlorate . . . . . 2 drams 8|0
Tincture of iron chloride . . . . . 2 drams 8|0
Camphor water . . . . . 6 ounces 200|0—M.
S.—One tablespoonful (15 c.c.) four times a day.

In very severe cases of ulcerative stomatitis, especially in such as develop epidemically, the patient should be isolated and the strictest antiseptic precautions carried out. The ulcerated surfaces should be brushed with silver nitrate (2 to 5 per cent.).

Patients who are taking mercury should use a potassium chlorate or potassium permangate or other antiseptic mouth wash. Upon the development of stomatitis the mercury must be withdrawn, the mouth wash continued, and a brisk purgative administered—whereupon most cases subside within a few days. Should ulcers develop they should be brushed with silver nitrate, and potassium chlorate may be given internally. Atropine is often useful in controlling the excessive salivation.

In thrush, in addition to the foregoing, especially cleanliness of the mouth and nursing nipples, alkaline mouth washes, such as lime water or sodium bicarbonate, to lessen the acidity of the secretions, and the withdrawal of starches and sugars from the dietary, are requisite. Locally, boric acid, borax, hydrogen peroxide, potassium permanganate, potassium chlorate, or sodium sulphite, may be used, and after the thrush spots have been removed the exposed mucous surface may be brushed with silver nitrate. In all varieties of stomatitis, but especially in thrush, attention must be directed to the diet, which must be nourishing, and to the general health, which should be improved by bitter tonics, iron, quinine, strychnine, cod-liver oil, etc.

GANGRENE OF THE CHEEK; NOMA.

(*Gangrenous Stomatitis; Cancrum Oris.*)

Noma is a rapidly spreading gangrene of the cheek, usually unilateral and frequently terminating fatally.

Etiology.—The disease occurs between the second and seventh years (although it may occur later), and in girls rather than boys, more especially in those debilitated by certain specific infective diseases, particularly measles (more than one-half the cases), pertussis, typhoid fever, scarlatina, etc. The disease is doubtless due to infection. Recently etiological significance has been attributed to a streptothrix described by Perthes and Seiffert.

Symptoms.—The disease usually begins insidiously, commonly during convalescence from measles or other infective diseases, and manifests itself as a discolored area which soon becomes necrotic, ulcerated, and malodorous, and about which there speedily develop, from within outward, a brawny induration and often widespread œdema, involving the side of the face and the neck. In mild cases the ulcer remains internal; but usually, sometimes within forty-eight or seventy-two hours, perforation, preceded by marked external discoloration, occurs. Sometimes the gangrene extends to the lips, chin, even the eyes and ears, and may involve the bone. The adjacent bloodvessels being thrombosed, hemorrhage is rare, but the gangrenous area is surrounded by an inflammatory zone. The regional lymph nodes are moderately enlarged, but local pain is not marked. The general symptoms are usually severe: high fever (104° F. or more), rapid pulse, extreme prostration, low muttering delirium, etc. Diarrhœa is common, and may be associated with colitis. Death usually ensues at the end of about a week, from toxemia or aspiration bronchopneumonia.

Diagnosis.—The diagnosis is usually unmistakable. Some confusion with malignant pustule (anthrax) may arise, but the previous history of the case is different; malignant pustule is more common in adults, begins on the outside of the cheek, and the anthrax bacillus may be detected in the local lesions and in the blood. Diphtheria may be readily differentiated by isolating the bacillus. Ulcerative stomatitis can scarcely be confused with gangrene, except perhaps in the earliest stages.

Prognosis.—The prognosis is bad, the mortality rate being 70 to 90 per cent.; a few patients recover with extensive cicatrization and deformity.

Treatment.—The treatment is usually unavailing. The disease is often far advanced before discovered. The gangrenous area must be removed preferably with the actual cautery, or with fuming nitric acid or the knife. The best results are likely to be achieved by the use of the actual cautery before gangrene has supervened. Subsequently strict antisepsis must be maintained, and the patient supported by concentrated nutritious food and stimulants. I have seen diphtheria antitoxin do temporary good in one case occurring in typhoid fever, from which the diphtheria bacillus was isolated.

DISEASES OF THE TONGUE.

Glossitis.—Glossitis, or inflammation of the parenchyma of the tongue, is a rare affection, although inflammation of the lingual mucous membrane is commonly a part of stomatitis.

Acute or parenchymatous glossitis results usually from injuries, or infections, or intoxications, such as mercury, the bites of insects, etc. The lesions, which may be unilateral, especially in the beginning, are those common to inflammation in general; usually they subside, but

they may go on to abscess formation. The *symptoms* consist of pain and extreme swelling of the tongue, which may even project beyond the teeth. All movements such as occur in speaking, chewing, swallowing, even breathing, are extremely painful, and hence restricted, at times almost impossible. Sloughing sometimes occurs. There may be moderate fever. The *treatment* consists in the local use of antiseptics, ice, cocaine, etc., general depurative measures, and surgical procedures, such as scarification or incision, should swelling be extreme or suppuration result.

Chronic glossitis, usually associated with chronic stomatitis, sometimes results from repeated acute attacks, but especially from continued irritation caused by carious and broken teeth, alcohol, tobacco, irritating food, etc. The tongue is swollen, reddened, hypersensitive, furrowed or fissured, and unnaturally smooth in circumscribed areas, due to atrophy of the papillæ. A condition in which the fissures and furrows are unduly marked, and the tongue irregular and ragged in appearance and painful (on account of superficial ulcerations), is spoken of as glossitis desiccans. The *treatment* consists of the avoidance of all irritating food and drink, the use of antiseptic mouth washes, brushing with silver nitrate (5 per cent.), and tonics.

Eczema.—Eczema of the tongue (geographical tongue, ringworm of the tongue, psoriasis of the tongue, tylosis of the tongue, wandering rash of the tongue) is a rare affection, of unknown and doubtless varying etiology, characterized by superficial hyperplasia and desquamation of the lingual epithelium. The lesions tend to heal in the centre and spread at the margins, giving rise to irregular circinate, map-like patches. The disease is chronic, and occasions only mild local symptoms, such as itching. The *treatment* consists in the use of tonics and antiseptic mouth washes, the local application of silver nitrate, tannic acid, alum, or potassium chlorate.

Leukoplakia.—Leukoplakia of the tongue (leukoplakia oris et buccalis, leukoma, ichthyosis of the tongue, keratosis, smoker's tongue) is a rather rare disease of unknown etiology, encountered usually in inordinate smokers, and characterized by the development of dense, white, somewhat elevated patches, varying in size and configuration, and situated usually on the tongue, rarely elsewhere on the oral mucous membrane. Usually smooth, the patches may become papillomatous, and even epithelioma may develop. The condition is chronic. The *treatment*, which is unsatisfactory, consists in the use of antiseptic mouth washes, and silver nitrate, chromic acid, or iodine. The use of tobacco must be prohibited, and to syphilitics appropriate treatment should be given. Should the patches become papillomatous, they should be removed.

Black Tongue.—Black tongue (nigrities) is a rare disorder, usually attributed to the action of a parasite, but by some observers to benign tumor growth, or to intentional staining, and is characterized by the development of a blackish-brown discoloration of the dorsum of the tongue, associated with elongation of the papillæ. The *treatment* con-

sists of attention to the general health, and the local use of silver nitrate, hydrogen peroxide, salicylic acid, boric acid, etc.

Macroglossia.—Macroglossia is a condition of enlargement of the tongue, sometimes found in akromegaly, myxœdema, and cretinism, and due usually to a lymphangioma, rarely to muscular hyperplasia. In extreme cases operative intervention may be called for.

Acute Œdema.—Acute œdema of the tongue, usually associated with œdema of the aryteno-epiglottidean fold, is a rare manifestation of urticaria. Though extreme, the swelling rarely lasts more than twenty-four hours. *Treatment* must be prompt—a drastic purge, scarification, or leeching of the tongue, and in extreme cases, tracheotomy.

Ranula.—Ranula is a cystic growth, situated on the under surface of the tongue, near the frenum, and due to dilatation of Wharton's duct, or to enlargement of a mucous gland.

DISEASES OF THE SALIVARY GLANDS.

Ptyalism.—Ptyalism (hypersecretion of the salivary glands, salivation) has already been mentioned as a symptom of mercurial stomatitis. It sometimes results also from the use of other drugs, such as gold, copper, iodine, tobacco, jaborandi, muscarin, etc. It occurs sometimes during pregnancy, rarely during menstruation; it is occasionally associated with disease of the pancreas, variola, and rabies; and it is not infrequent in certain diseases of the nervous system, such as hysteria, insanity, and divers diseases of the pons and medulla. The amount of saliva is sometimes very great (four or five liters), exceeding many times the normal (one to one and one-half liters). The *treatment* is that of mercurial stomatitis.

Xerostomia.—Xerostomia (hypersecretion of the salivary glands, dry mouth, aptyalism), deficiency or suppression of the salivary and buccal secretions, is a rare condition of unknown etiology (although usually said to be a neurosis), occurring especially in neurotic women, in whom it follows sudden fright or shock. The mouth is unusually dry; sordes collect on the teeth and gums; the tongue may become fissured; and the breath is fetid. The condition is to be distinguished from the dry mouth of fever conditions, and that associated with the mouth-breathing of adenoids. *Treatment* is unsatisfactory, although oily applications locally, pilocarpin lozenges ($\frac{1}{15}$ grain, 0.004 gram) slowly dissolved in the mouth several times daily, and galvanism locally, may be productive of good.

Parotitis.—Inflammation of the parotid gland may develop, (1) as an acute specific infective disease—mumps (page 260); (2) as a symptomatic affection—symptomatic parotitis, parotid bubo; and (3) as a chronic affection.

Acute symptomatic parotitis results from: (1) Infections, such as typhoid fever, typhus fever, scarlet fever, pneumococcic infection, strep-

tococcic infection (pyemia, erysipelas, etc.); (2) disease or injury of the abdominal or pelvic organs; and (3) in association with inflammation of the facial nerve. In most cases the disease is infectious, the infective agent being carried through the blood or by way of the parotid (Stenson's) duct. The inflammation is unilateral (in contradistinction to mumps, which is bilateral). The gland is swollen, painful, and tender, especially on opening the mouth, and in 50 per cent. of the cases proceeds to suppuration. The general *symptoms* are moderate, as a rule —malaise, headache, and slight fever. The *treatment* consists of general depurative measures, and the local application of ice, leeching, lead water and laudanum, etc. In the event of suppuration, surgical measures are called for.

Chronic parotitis rarely follows mumps, or the continued use of mercury, potassium iodide, and lead. It has been observed in chronic nephritis and in syphilis. Rare cases of chronic painless symmetrical enlargement of both parotid and lacrymal glands have been observed by Mikulicz (Mikulicz's disease) and others.

Gaseous Distention.—Gaseous distention of the parotid duct and gland has been observed in glassblowers and musicians.

Calculi.—Salivary calculi occasionally form in the parotid duct, and may give rise to distention of the duct and enlargement of the parotid gland.

LUDWIG'S ANGINA.

Ludwig's angina (angina Ludovici, cynanche gangrænosa) is a rare phlegmonous cellulitis of the floor of the mouth and of the neck, commonly due to the streptococcus, and occurring usually as a complication of certain infective diseases, especially scarlet fever and diphtheria. It occasionally follows local trauma, and rarely occurs as an apparently primary affection. The *symptoms*, which develop and progress with great rapidity, consist of pain, tenderness, redness, marked swelling, and induration, arising usually in the neighborhood of the submaxillary gland on one side, and speedily involving the floor of the mouth, the entire neck, and even the upper part of the chest wall. The swelling of the tissues of the neck and an associated œdema of the glottis frequently give rise to an extreme, alarming, and sometimes fatal dyspnœa (asphyxiation). Extensive sloughing and gangrene are common. The general symptoms are those of profound sepsis (toxemia). Although the outlook is extremely grave, prompt surgical intervention, supportive measures, and antistreptococcic serum may result in cure.

DISEASES OF THE FAUCES, TONSILS, AND PHARYNX.

ACUTE TONSILLITIS.

(*Acute Faucitis, Tonsillitis, and Pharyngitis; Sore Throat; Angina.*)

Etiology.—Acute tonsillitis, usually associated with faucitis and pharyngitis, may occur at any age, but is most common in adolescents, especially rheumatic and scrofulous subjects, and in those debilitated from any cause. It is especially prevalent in the spring and the early fall, and its development is much favored by insanitary surroundings. The exciting causes are infectious, toxic, mechanical, or thermic irritants. Infectious angina may be primary or secondary. Most primary cases bear an undoubted relation to exposure or "catching cold." Contrary to former opinions, this is now believed to act by reducing the resistance of the tissues, thus permitting the easy operation of bacteria, of which the most important etiologically are the streptococcus, the staphylococcus, the pneumococcus, the influenza bacillus, and the pneumobacillus (Friedländer). An angina is also an initial manifestation in certain specific infective diseases, such as diphtheria, scarlet fever, measles, etc. Secondary infectious anginas are common in other infective diseases, such as variola, varicella, typhoid fever, tuberculosis, syphilis, etc. A form of infectious angina sometimes prevails epidemically, especially in institutions, etc. Toxic anginas result from the inhaling of irritant vapors, such as acids and alkalies; from the excessive and prolonged use of alcohol, tobacco, etc.; and from the medicinal and other use of mercury, the iodides, etc. Inhalation of hot vapors and the ingestion of too hot food and drink sometimes provokes acute angina. An acute angina, especially acute tonsillitis, is frequently the initial manifestation of a systemic infection, the infectious agent gaining access to the general circulation by way of the tonsils—thus rheumatic fever, acute endocarditis, chorea, influenza, streptococcic infection, tuberculosis, syphilis, etc. Recurrences are common.

Pathology.—The lesions may involve the fauces, tonsils, and pharynx, singly or conjointly, and equally or unequally. In most cases all are more or less involved. In mild cases, the faucial and pharyngeal mucous membrane and submucous tissues are injected and swollen, and later covered with a slight amount of mucous or mucopurulent exudate (catarrhal faucitis, tonsillitis, or pharyngitis; sore throat). In other cases the tonsils present scattered yellowish-white spots—distended follicles filled with desquamated epithelium, leukocytes, granular debris, and bacteria (follicular tonsillitis). Usually the exudate can be squeezed from the follicles, but occasionally several adjacent follicles being distended, the tonsil appears to be covered with a pseudomembrane, much simulating diphtheria. Occasionally herpes develop (herpetic tonsillitis).

In more severe cases the inflammation involves the substance of the tonsil, and usually one tonsil more than the other (parenchymatous tonsillitis). The affected tonsil becomes enlarged, sometimes enormously swollen, and projects far beyond the median line, rendering phonation, deglutition, articulation, and even respiration, extremely difficult. Usually the lesions soon proceed to suppuration (suppurative tonsillitis, quinsy), and, should surgical intervention be delayed, the abscess ruptures, usually through the free surface of the tonsil, rarely through the soft palate. At times the pus may find its way into the tissues of the neck or mediastinum, and may even perforate the vessels of the neck. Rarely more or less extensive sloughing and subsequent ulceration of the tonsil and pseudomembranous formation occurs (ulcerative tonsillitis). The lesions may extend to the uvula, soft palate, and even the posterior pharyngeal wall, and, when occurring as a primary affection, they are to be distinguished from diphtheria only by the absence of the diphtheria bacillus. Streptococci and staphylococci are usually present in abundance.

Symptoms.—Clinically one may distinguish (1) catarrhal, (2) follicular, and (3) suppurative or phlegmonous tonsillitis.

Acute catarrhal tonsillitis, usually associated with faucitis and pharyngitis, is frequently sudden in onset, with chilliness, sometimes a distinct chill, and moderate fever. Vomiting may occur in children. An initial sense of dryness and irritation of the throat are soon succeeded by distinct pain, especially upon swallowing, and a harsh, dry cough. Upon inspection, the tonsils, fauces, and pharynx are swollen, œdematous, congested, and covered with a thin layer of mucopurulent matter, which at the end of thirty-six or forty-eight hours is usually sufficient to give rise to a small amount of tenacious sputum. The general symptoms are mild, and the lesions subside within four or five days.

Acute follicular (*lacunar*) *tonsillitis* is a more severe infection. The onset is frequently quite abrupt, with pronounced chill and high fever—103.5° F. in adults, and frequently 105° F. in children. The faucial pain and tenderness and the difficulty in swallowing are usually marked, and there is sometimes tenderness at the angles of the jaw, on account of moderate swelling of the regional lymph nodes. On inspection, in addition to catarrhal alterations of the faucial mucous membrane, the tonsillar crypts are distended with a characteristic yellowish exudate, which, upon pressure, may be generally forced out. The constitutional symptoms are often quite marked, and consist of high fever, headache, general neuromuscular pains, depression, loss of appetite, constipation, etc. A trace of albumin in the urine is not uncommon. The disease usually runs its course in six or eight days and terminates in recovery, but endocarditis, pericarditis, pleuritis, erythema nodosum, etc., may occur as sequels.

Acute suppurative tonsillitis (phlegmonous tonsillitis, parenchymatous tonsillitis, tonsillar and peritonsillar abscess, quinsy) does not differ in its initial local and general symptoms from those just mentioned, although

they are usually severe. Pain is pronounced, deglutition is extremely difficult, and phonation often practically impossible. On examination, the tonsil and adjacent tissues are found extremely swollen, congested, and œdematous. Not infrequently the tonsils meet in the median line; usually one is more inflamed than the other, and may project beyond the median line, displacing the markedly œdematous uvula. The glands at the angles of the jaw are usually swollen and tender, and there may be a notable fulness in the neck. The oral secretions are increased, and usually dribble from the mouth. Moderate leukocytosis is usually present. In mild cases, resolution takes place, but in perhaps one-half of the cases, at the end of forty-eight or seventy-two hours, fluctuation may be detected, and, if the tonsil be not incised, a yellowish spot of "pointing" soon appears and the abscess ruptures. Usually this is attended by great relief, and the expectoration of the pus is followed by speedy subsidence of the inflammatory phenomena. Rarely, however, the rupture occurring during sleep, for instance, may lead to suffocation; or the purulent matter may burrow into the tissues of the neck, and find its way externally through the skin, or even perforate the great blood-vessels of the neck; or œdema of the glottis may occur. During the height of the disease the general symptoms continue well marked, and the temperature remains high (104° F.), the pulse frequent (120 to 130 per minute), and evidences of toxemia are found in concentration of the urine, slight albuminuria, prostration, perhaps delirium, etc. The lesions usually subside within ten days.

Diagnosis.—The diagnosis is usually unattended by special difficulties. It is sometimes difficult to distinguish follicular tonsillitis from diphtheria. In many cases the diagnosis rests entirely upon the detection of the diphtheria bacillus; in some cases, however, one may distinguish follicular tonsillitis by the fact that the exudate is yellowish, patchy (corresponding to the follicles), separated by intervening areas of congested red tonsillar tissue, does not tend to spread beyond the tonsils, may be removed without leaving a bleeding surface, and does not tend to reform. In diphtheria, on the other hand, the exudate is usually grayish in color, uniform and widespread, tends to extend beyond the tonsils, and when removed tends to reform.

Prognosis.—The prognosis is good. Most cases result in recovery, although recurrences are common, and hypertrophy of the tonsils frequently results.

Treatment.—In all but the mildest cases the patient should go to bed and curtail as much as possible all movements of the throat, such as occur, for instance, in talking, swallowing, etc. The diet should be fluid. A thorough evacuation of the bowels by a generous dose of calomel (5 grains, 0.3 gram), allowed to dissolve slowly on the tongue and to mix with the oral secretions (whereby it may exert a local antiseptic action), and followed by a saline is often of value. The course of the disease may sometimes be shortened, in some cases apparently aborted, by the early administration of sodium salicylate or benzoate in large

doses, 15 grains (1 gram), four or five times a day for the first twenty-four or thirty-six hours. In other cases, tincture of iron chloride (30 minims, 2 c.c.), quinine (2 grains, 0.13 gram), with glycerin and water, every four hours for thirty-six hours, seems to exert a very beneficial effect. Neither form of treatment, however, is of much value if begun when the disease is fully developed. In the case of children, full doses of aconite have been much praised. Locally, alkaline and mild antiseptic solutions may be used—such as Dobell's solution, potassium permanganate (2 per cent.), thymol, alum, etc., or one may use the old-fashioned ammoniated tincture of guaiac, in water, as a gargle; or lozenges of guaiac may be dissolved slowly in the mouth. In some cases good results attend the local application to the tonsils of aspirin, silver nitrate (25 per cent.), or tincture of iron chloride, glycerin, and water. The comfort of the patient may also be promoted by ice locally to the throat and dissolved in the mouth, by the inhalation of medicated steam (with compound tincture of benzoin), and by the administration at night of a Dover's powder (10 grains, 0.6 gram).

In extreme swelling, especially of one tonsil, such as portends suppuration, early scarification will relieve the local pain and distress. Incision should be practised as soon as fluctuation is apparent, even suspected. Cocaine (2 to 5 per cent.), menthol (2 per cent. in albolene), and hot, rather than cold, applications are also of service.

During convalescence, the patient requires a nourishing diet and tonics, such as iron, quinine, and strychnine; or Basham's mixture and strychnine. Should the tonsil remain permanently enlarged (chronic tonsillitis), appropriate treatment must be instituted.

CHRONIC TONSILLITIS.

(*Enlarged Tonsils; Hypertrophied Tonsils; Adenoid Vegetations; Chronic Nasopharyngeal Obstruction; Mouth-breathing; Aprosexia.*)

Chronic tonsillitis, in which the tonsils frequently become markedly enlarged, is usually associated with hyperplastic alterations of the adenoid tissue of the pharynx (pharyngeal tonsil, Luschka's tonsil). The condition is of extreme importance, since it not only occasions nasopharyngeal obstruction and consequent mouth-breathing, but it exerts considerable deleterious influence on the physical and mental development (aprosexia) of its victims—mostly children.

Etiology.—Hypertrophy of the tonsils and pharyngeal adenoids are most common between the ages of five and fifteen years. They occur in both sexes, but probably affect boys more than girls, especially children of tuberculous and syphilitic parents, and those who are poorly nourished and live amidst unhygienic surroundings. The affection often dates from an acute attack of tonsillitis, simple, diphtheritic, or scarlatinous.

Pathology.—The lesions are those of true hypertrophy of the tonsillar constituents—in most cases, especially of the lymphoid tissue, in other

cases, of the stroma. In the first the tonsils are much enlarged (twice or thrice the normal size), irregular and rough on the surface, and exhibit distended and ruptured follicles. Although both tonsils are usually involved, the process may be asymmetrical. In the event of marked hyperplasia of the stroma, the tonsils are smaller and much firmer. The pharyngeal adenoids usually project from the pharyngeal vault, growing from the fossa of Rosenmüller—a depression on the posterior pharyngeal wall between the Eustachian orifices. They consist of adenomatous papillomatous growths, and vary in size from 2 to 3 mm. to 2 to 3 cm.; they are reddish in color, very vascular, and moderately firm. There is usually an associated chronic nasal catarrh; the hard palate is often unusually high, and the alveolar arch small.

Symptoms.—Minor forms of chronic tonsillitis (with hypertrophy) may exist with no noteworthy symptoms. Sooner or later, however, there is interference with nasal respiration, and mouth-breathing develops. Although present during the day, this is noticed especially during the night, when the breathing is loud and snoring and frequently interrupted—whence night terrors arise. Coughing is sometimes provoked by lying down. The mouth is dry and the breath foul from decomposition of the exudate in the tonsillar crypts. Characteristic physical and mental changes soon occur. In general the child is of stunted growth—attributed by some authors to the embryological relationship between the tissues of the pharynx and the pituitary body. The mouth is usually open, the expression becomes vacant, the bridge of the nose depressed, the malar prominences ill developed, the lips thick, the hearing defective (on account of obstruction to the Eustachian orifices, or extension of disease along the Eustachian tubes), the voice nasal, and articulation indistinct and sometimes stammering. The so-called pigeon-chest, or chicken-chest, frequently develops, especially in rachitic children. In some children with recurring asthmatic attacks a typical barrel-shaped or emphysematous chest may develop. Rarely a funnel-chest (Trickterbrust) has been observed. In advanced cases, marked changes in mentality occur. The child is listless, backward, forgetful, and seems unable to control his attention (aprosexia). He is sometimes morose and irritable, and complains of headache. He is usually weakly, anemic, and has a poor appetite, and is prone to recurring attacks of follicular tonsillitis, diphtheria, scarlet fever, etc.

Diagnosis.—The affection is often disclosed by the facial, physical, and mental characteristics of the child. The tonsillar changes are quite apparent upon inspection of the throat, but the detection of the pharyngeal lesions, which may exist without noteworthy tonsillar changes, may require a rhinoscopic or digital examination.

Prognosis.—Although the disease scarcely endangers life, it is of much significance in shaping the physical and mental characteristics, and the bad habits and general stunting that it occasions are sometimes not outgrown in later life.

Treatment.—In mild cases of chronic tonsillitis the local application of iodine and glycerin, Lugol's solution of iodine and potassium iodide, tincture of iron chloride and glycerin, tannin, silver nitrate, etc., may prove useful. In general, however, the wise course is to remove the enlarged tonsils with a tonsillotome or galvanocautery, or by careful dissection. In all cases the pharyngeal adenoid should be immediately removed, with a curette or a finger, even should the child as yet not present the facial and mental characteristics of the disease, which will surely develop in the course of time. Attention to the general health, by suitable dress, fresh air, nutritious food, tonics, cod-liver oil, syrup of iron iodide, etc., is imperative.

PHARYNGITIS.

Acute pharyngitis, occurring independently of acute tonsillitis, is unusual, but is sometimes the lesion in an ordinary "sore throat." The symptoms and treatment are those outlined under acute tonsillitis.

Chronic pharyngitis in children is associated with hypertrophy of the tonsils. In adults it is usually part of a chronic nasopharyngeal catarrh; it follows repeated acute attacks, and occurs especially in alcoholic subjects, inordinate smokers, and in those who use the voice much and injudiciously, such as clergymen (clergymen's sore throat), public speakers, singers, hucksters, etc. Several varieties have been distinguished: (1) Chronic, simple, or hypertrophic nasopharyngeal catarrh; (2) chronic atrophic pharyngitis (pharyngitis sicca), usually associated with atrophy of the nasal mucous membrane; and (3) follicular or granular pharyngitis. All three varieties are sometimes irregularly associated. There is usually relaxation of the faucial and pharyngeal mucous membrane, marked venous enlargement, and a varying amount of hyperplastic lymphoid tissue about the mucous follicles. The *symptoms* are a constant sense of dryness and irritation in the throat, interfering with speaking and singing, and more or less persistent "hawking." In the *treatment* it is important to correct the cause—prohibit smoking and alcohol, and the too prolonged and vigorous use of the voice, etc. The subjective symptoms may be relieved by different mild antiseptic lotions or lozenges, and by the local use of silver nitrate (2 to 5 per cent.). Areas of glandular hypertrophy should be removed by the galvanocautery. The general health should be maintained.

ULCERATION OF THE PHARYNX.

Ulceration of the pharynx occurs under a variety of circumstances. The only noteworthy symptom is pain, especially upon deglutition, but it varies much in different cases. *Follicular ulceration* is common in chronic catarrh—the ulcer being small and superficial. *Syphilitic ulcers*

are usually associated with mucous patches or evidences of syphilis elsewhere on the body; they are comparatively painless, superficial (unless the consequence of softening of a gumma), fairly regular in outline, and respond readily to antisyphilitic treatment. *Tuberculous ulcers* are common in the later stages of tuberculosis, are exquisitely painful, grayish yellow in color, and irregular in outline. Rarely the infectious atrium in tuberculosis may be about the tonsil and take the form of an ulcer. In diphtheria and scarlet fever, superficial ulceration is not uncommon. Ulceration occurs also in lupus, in typhoid fever, variola, carcinoma, etc.

Acute infectious phlegmon of the pharynx, is a term used by Senator to designate a rare condition in which the pharynx and the tissues of the neck in general become the seat of an intense inflammation that rapidly progresses to suppuration. The swelling and inflammatory œdema may be so marked as to interfere with respiration. The general *symptoms* are those of sepsis. A suppurative or phlegmonous pharyngitis may also follow trauma, or suppurative tonsillitis, or be associated with erysipelas. The *treatment* is surgical and supportive.

Retropharyngeal abscess is a suppurative inflammation of the postpharyngeal connective tissues. It is common in childhood, especially before the end of the second year, but it occurs also in adults. In children it frequently arises as an apparently primary infection, the infectious agent gaining access through any of the lymphoid tissues of that region; it sometimes follows diphtheria and scarlet fever. In children and in adults it may follow caries of the vertebræ. The *symptoms*, although referred to the throat, are not characteristic. The *diagnosis* is quite apparent upon inspection and digital examination of the throat, although it may be overlooked on account of the bulging of the postpharyngeal wall being high up. The *treatment* consists of free evacuation of the pus, which should be done promptly, or the patient may suffocate.

Elongation of the uvula, although frequently imaginary and occasionally congenital, sometimes results from repeated attacks of faucial inflammation, and may occur in conditions of malnutrition. By irritating or tickling the throat on lying down, it may excite a reflex cough, or rarely spasm of the glottis. The *treatment* consists in excision, should the measures indicated under chronic tonsillitis not suffice.

The lingual tonsils sometimes share in inflammatory and other diseases of the throat. Occasionally they are markedly involved, either alone or in association with disease of contiguous tissues, and they occasion pain and a feeling of fulness and discomfort at the base of the tongue, and a reflex cough. Occasionally they become hypertrophied and interfere with the actions of the epiglottis. The *treatment* is similar to that of tonsillitis.

DISEASES OF THE ŒSOPHAGUS.

Acute Œsophagitis.—Etiology.—Œsophagitis may be due to infective, toxic, mechanical, or thermic irritants. Infective œsophagitis occurs in a mild or catarrhal form in many of the infective diseases, and at times as an extension of inflammation from the mouth and throat. Severer pseudomembranous forms sometimes occur in diphtheria, thrush, pneumonia, pyemia, etc., a pustular form may occur in smallpox. Toxic œsophagitis may follow the ingestion of strong acids and alkalies; a chronic form is sometimes observed in those that use much alcohol or tobacco. Mechanical œsophagitis may result from the irritation produced by foreign bodies. Thermic œsophagitis may follow the taking of overheated food or drink.

Pathology.—The lesions vary much in different cases, and may be described as catarrhal, ulcerative, pseudomembranous, pustular, phlegmonous, and gangrenous. They exhibit no characteristics that distinguish them from similar lesions occurring elsewhere, except that rarely in the pseudomembranous form casts of the œsophagus may be formed and perhaps expelled.

Symptoms.—Mild œsophagitis may occasion no symptoms. Usually there is substernal pain, which is much aggravated by swallowing; in some cases with marked lesions, however, there may be little or no pain except on swallowing. In severe forms due to corrosive poisons, swallowing may be impossible. In some cases, especially in the event of a foreign body being present, attempts at swallowing occasion spasm and regurgitation of food. In severe cases, mucus, sometimes blood and pus, are ejected, and the patient emaciates. A stricture usually follows the œsophagitis due to corrosive poisons.

Treatment.—The treatment is unsatisfactory. In the mild cases demulcent drinks and ice water may be given. Silver nitrate in solution and weak solutions of cocaine may prove beneficial, especially in allaying pain. In severe cases, feeding by the mouth should be substituted by enemas.

Chronic Œsophagitis.—Chronic œsophagitis may result from an acute attack, or from the long-continued action of alcohol, tobacco, and other irritants. The mucous membrane becomes thickened and sometimes markedly rugous. The *symptoms* are more or less substernal pain, especially on swallowing, and perhaps regurgitation of food and mucus. The *treatment* is similar to that of acute œsophagitis.

Ulceration.—Ulceration of the œsophagus may occur: (1) As a part of acute œsophagitis; (2) occasionally in cachectic states; (3) in typhoid fever, when it may be followed by cicatricial stenosis, and in syphilis; (4) in malignant disease; (5) in consequence of pressure, as by an aneurysm; and (6) as the counterpart of the round or peptic ulcer of the stomach. The *treatment* of the condition, which, however, is rarely recognized, is that of a causative factor, or of gastric ulcer.

Varices.—Varices of the œsophagus, usually situated at the lower end of the gullet, and sometimes of enormous size, may occur in cirrhosis of the liver and in chronic heart disease. The mucous membrane is the seat of chronic inflammation, and the patient may eject considerable mucus, but usually the condition is not recognized until a large, and sometimes fatal, hemorrhage occurs.

Rupture.—Rupture of the œsophagus rarely results from the excessive strain of prolonged vomiting, or from weakness of the wall due to an ulcer.

Dilatation.—Dilatation of the œsophagus is rare. Exceptionally it is primary—when it is usually congenital, but it is said to have developed in consequence of weakness and dilatation of the wall. In most cases the dilatation is secondary to, and develops above, a constriction. The dilatation may be fusiform or cylindrical. The chief *symptoms* are dysphagia, regurgitation of food, and loss of weight from insufficient nourishment. The chief aid in the diagnosis results from radiographic examination after the injection of bismuth or iron oxide pastes. When the patient can no longer get food into the stomach, he may be assisted with the stomach tube. Eventually rectal enemas and possibly gastrostomy will be demanded.

Diverticula.—Diverticula of the œsophagus (pharyngoceles) are of three kinds: (1) Congenital. (2) Pressure or pulsion diverticula, which are rare, arise from pressure within the œsophagus (the swallowing of large boluses of food), are usually situated on the posterior wall of the pharynx at the junction of the pharynx and œsophagus, are rarely long, and occur usually in adults. (3) Traction diverticula, which are not so rare, arise from external traction of the œsophageal wall (contraction of a cicatrix following inflammation of a mediastinal lymph node), are usually situated on the anterior wall of the œsophagus at the bifurcation of the trachea, are usually quite short, and occur usually in children. Traction diverticula rarely occasion symptoms. Pressure diverticula when large may give rise to dysphagia, fetor of the breath (on account of decomposition of retained food particles, etc.), nausea, vomiting, and rarely a bulging on one side of the neck. The diagnosis is best made by radiographic examination after the injection of bismuth or iron oxide pastes.

Spasm.—Spasm of the œsophagus (œsophagismus, spasmodic stricture of the œsophagus) is observed in neurotic, especially hysterical, subjects, and is more common in women, although it occurs in men. It is sometimes present during pregnancy, epilepsy, chorea, and hydrophobia. The attack may develop suddenly, being provoked by violent emotional or other excitement. The chief complaint is of inability to swallow food, sometimes even liquids being rejected. An œsophageal bougie may be passed—sometimes immediately, sometimes after waiting a little for the spasm to relax and exerting slight pressure. This of itself may effect a lasting cure. The condition is readily recognized, as a rule, but in elderly subjects care must be taken not to overlook malignant stricture. The neurotic basis of the condition demands treatment.

Paralysis.—Paralysis of the œsophagus occurs in certain diseases of the nervous system, functional (hysteria) as well as organic (bulbar paralysis, general paresis, diphtheritic paralysis, etc.). The main *symptom* is dysphagia; usually also there are regurgitation of food and emaciation.

Stenosis.—Stenosis (organic stricture) of the œsophagus may be congenital or acquired. *Acquired stenosis* may be due to: (1) Swelling and thickening of the œsophageal wall, such as is occasioned by inflammatory (especially phlegmonous) swellings, tumors, etc.; (2) cicatrization of ulcers produced by corrosive poisons, syphilis, typhoid fever, diphtheria, and the peptic or round ulcer, etc.; and (3) external pressure produced by an aneurysm, pericardial effusion, mediastinal tumors, enlarged lymph nodes, goitre, etc. Stenosis is more common in males than in females. It may occur anywhere throughout the œsophagus, but is more common in the lower third or the upper third. It may be moderate or extreme, and annular or vertical, and if vertical, it may involve a small portion or almost, if not quite, all of the œsophagus. The *symptoms* consist of gradually increasing dysphagia and regurgitation of food. At first there is perhaps mere discomfort on swallowing, but later sometimes inability to swallow even liquids. Regurgitation of food follows immediately, or after a short time, depending upon the situation of the stenosis. When the stenosis is near the stomach and the œsophagus is dilated, the delay in regurgitating the food may suggest that it has been vomited, but its alkalinity and the absence of gastric odor disprove this. Hunger and thirst are usually marked and anemia and emaciation soon develop. The dilatation and degree of the stenosis may be determined by the use of radiography or an œsophageal bougie, or by œsophagoscopy. It is said also that in the event of stricture, auscultation over the eighth or ninth thoracic vertebra will reveal a deglutition murmur (when the patient swallows), longer than the normal seven seconds. The *treatment* in benign cases consists in gradual dilatation with bougies. In some cases a tube has been inserted and left in situ indefinitely, the patient being fed through it. Gastrostomy may be resorted to in malignant cases, as well as in some that follow corrosive poisons.

Carcinoma.—Carcinoma of the œsophagus occurs especially in men beyond the fortieth year. It is usually situated in the middle and lower thirds, especially at the bifurcation of the trachea. It usually develops from the surface epithelium (squamous-cell variety), rarely from the cells of the mucous glands (adenocarcinoma). It usually encircles the œsophagus, occasioning an annular constriction, and it may extend throughout a considerable portion of the œsophagus. Above the growth the œsophagus usually dilates and its wall hypertrophies. As a rule, the carcinoma soon ulcerates, and a considerable portion of the necrotic tissue may be vomited, thus increasing the lumen again. Metastasis to the regional lymph nodes and to the nodes just above the clavicle, as well as extension to adjacent tissue (the mediastinum, lung, pleura, pericardium, vertebræ, etc.), are common, and perforation of

a bronchus, or the aorta, etc., is not unknown. The *symptoms* consist of substernal pain or discomfort, gradually increasing dysphagia, regurgitation of food, and progressive emaciation with the development of the cancerous cachexia. The regurgitated material is frequently fetid, and may contain blood and pus from ulceration, and sometimes portions of the carcinoma itself. The development of certain pressure symptoms, such as paralysis of the recurrent laryngeal nerve, oculopupillary phenomena, or a bronchial cough in association with œsophageal symptoms, are of extreme diagnostic importance. The affection must be differentiated from pressure from without, as by an aneurysm, and until this be excluded the œsophageal bougie should not be used. The bougie may dislodge a portion of the growth, examination of which will facilitate the diagnosis. The *treatment* is merely supportive and palliative, as the disease is usually fatal within a year. Gastrostomy is sometimes of service.

DISEASES OF THE STOMACH.

Pathological Physiology.—The stomach, the most widely dilated portion of the digestive tract, has an average capacity in the adult of about 1600 c.c. (Ewald), with a normal maximum of about 2000 c.c. It is designed for the reception, at one time, of a considerable amount of food and to effect certain digestive and other changes in this food so as to render it suitable for further alterations in the intestine. The important changes, aside from the digestive, that the food undergoes in the stomach, comprise its conversion into a more or less fluid consistency, its being brought to a proper temperature (since the intestine is more sensitive to temperature variation, especially cold, than the stomach), and its partial sterilization by free hydrochloric acid.

The chief functions of the stomach are secretory and motor in nature. The secretory activity is expressed in the gastric juice, which results from the influence of psychic and chemical stimuli. Mechanical stimuli, as a rule, induce only an increased secretion of alkaline mucus, which is designed primarily to protect the lining of the stomach from mechanical insults; the frequent repetition of these insults (bolting of large masses of unchewed and non-insalivated food) by irritating the stomach and impeding digestion, may ultimately lead to chronic gastritis. The psychic stimuli that promote the secretion of gastric juice comprise the pleasurable sensations of the sight, odor, and anticipation of food, often enhanced by agreeable companions and other environment—all of which suffice, even when the stomach is empty, to inaugurate a flow of gastric juice—of appetite juice as Pawlow has expressed it. The chemical stimuli comprise the chemical constituents of certain foods and condiments, such as *hors d'œuvre,* meat extracts represented in soups, other proteins, etc. The customary arrangement of the courses at dinner thus finds justification in the normal physiological activities

of the stomach. Starches and other carbohydrates (later courses at dinner) do not promote any noteworthy flow of gastric juice; they are, therefore, indicated in cases of excessive gastric secretion, whereas the aforementioned excitors of gastric juice are indicated in cases of deficient secretion; and it is a matter of common knowledge that an invalid with poor appetite can be made to eat and to digest by such environment as well arranged and tastefully served food. The secretion of gastric juice begins usually before food has entered the stomach and it continues, as a rule, for an hour or two thereafter, whereupon it gradually lessens. Variations in this, however, are common, being due to individual peculiarities, the amount and character of the food, etc., and in diseased states too much or too little gastric juice may be secreted—hypersecretion and hyposecretion, respectively.

The normal gastric juice is a thin, colorless (or almost colorless) fluid; it has a characteristic odor, is strongly acid in reaction, and has a specific gravity of 1002 to 1003. Its essential constituents are hydrochloric acid, pepsin (a proteolytic enzyme), rennin (lab-ferment, a milk-curdling enzyme), and, in small amount, lipase (a fat-splitting enzyme); in addition, mucin, inorganic salts, and water are found. The hydrochloric acid is a product of certain so-called border or cover cells present in the fundic glands, which are especially numerous in the midportion or prepyloric region of the stomach. Normally hydrochloric acid is present in the gastric juice in the proportion of 0.3 per cent., although it may reach higher values. Expressed in terms of the amount in cubic centimeters of decinormal sodium hydrate solution required to neutralize it (the method commonly employed in clinical work), the free hydrochloric acid content of the gastric juice normally varies between 20 and 40 per hundred (which figures multiplied by 0.00365 give the actual hydrochloric acid percentage). The total acidity of the gastric juice, that is, the free hydrochloric acid, plus the combined hydrochloric acid, acid salts, and organic acids, normally varies between 30 and 50, with 60 as a normal maximum. These values may be notably increased or decreased in disease, and hydrochloric acid may be entirely absent (see secretory disorders).

Pepsin, the proteolytic enzyme of the stomach that breaks down proteins into albumoses and peptones, is active only in an acid medium; and since pepsinogen or propepsin is activated by the hydrochloric acid, the acidity of the gastric juice and peptic activity usually run parallel. At all events if the hydrochloric acid of the gastric juice is normal or increased, it is usually unnecessary to determine the peptic activity. In chronic gastritis with subacidity the peptic activity may be lessened; but even in this event if the mobility of the stomach remains effective the patient may suffer no noteworthy ill consequences. Rennin (lab-ferment, milk-curdling enzyme) is sometimes found deficient or absent in achylia gastrica, certain forms of chronic gastritis, carcinoma, etc. Lipase (fat-splitting enzyme) also may be found absent in like circumstances.

As food is taken into the stomach, it is received in the fundus, the chief function of which is to act as a reservoir, gradually to mix the gastric juice with the food, and to pass the mixed food on to the pyloric portion of the stomach. The later portions of a large meal are received into the mid-portion of the mass of food already in the stomach; and since the movements of the fundus are not very active, contact with the gastric juice is only gradually effected and salivary digestion thus may continue for some time. The pyloric end is separated from the rest of the stomach by a more or less well-defined muscular band, which serves to prevent mixing of the food in these two portions of the organ. Food is gradually received into the pyloric portion through the intermediation of a pre-pyloric (pre-antral) or middle portion, the seat of regularly recurring contractions (every fifteen or twenty seconds). In the pylorus the muscular movements are much more active than elsewhere; here the food is finely comminuted, converted into a more or less fluid consistency, intimately comingled with the gastric juice, and passed on to the duodenum. The cause of the movements of the stomach has not been definitely determined; they have been ascribed to the hydrochloric acid, but since they occur in the entire absence of hydrochloric acid, it is not unlikely that they are due to the presence of the food itself; they seem to be myogenic in origin, but they are doubtless under the control of the nervous system. The intermittent opening of the pylorus and the discharge of the gastric contents into the duodenum also have been attributed to the hydrochloric acid, and the closing, to the alkalinity of the duodenum (Cannon). Doubtless this is a factor, but not the sole factor, since it occurs in the absence of hydrochloric acid and hyperchlorhydria appears to be an important factor in producing pylorospasm. Disorders of the motility of the stomach are discussed under a separate heading.

SYMPTOMATIC DISORDERS OF THE STOMACH.

Disorders of the functions of the stomach, notably epigastric pain, nausea, and vomiting, are the natural consequence of definite anatomical lesions of the stomach of the most diverse nature, and as such they are usually recognized and correctly referred to a more or less obvious cause. In other cases, they are manifestations of disease of adjacent or other abdominal viscera, especially the gall-bladder, the duodenum, the pancreas, the vermiform appendix, and the intestine (obstruction); in other cases they are due to, or are part and parcel of, some general neurosis, such as hysteria or neurasthenia; whereas in other cases, the gastric symptoms seem to be due entirely to some perversion of the functional activity of the gastric nervous mechanism, and are not associated with discoverable anatomical lesions; in other words, the condition is a neurosis.

The Appetite.—*Loss* or *impairment of appetite* (anorexia) is a symptom of the most diverse disorders. It is common in all diseases of the stomach,

although, on the contrary, even serious diseases of the stomach may exist with little or no impairment of the appetite. Anorexia is a characteristic symptom of the chronic alcoholic subject (chronic gastritis), and is common in many dyspeptics who, for one reason or another, have gradually but progressively restricted their dietary. Furthermore, the appetite is impaired in all fever states, in debilitated conditions (carcinoma, tuberculosis, prolonged suppuration), in anemic states (chlorosis, pernicious anemia), in nervous disorders, such as hysteria, and following grip, worry, excitement, etc., and it may itself be a neuroses (nervous anorexia). *Increase of appetite* (boulimia) is common in convalescents from acute infectious diseases, especially typhoid fever; it occurs in certain cases of diabetes; in pertussis (on account of the large amount of food often lost by vomiting); in certain cases of gastritis (after the anorexia passes away); and it is a manifestation of certain neurotic conditions (see sensory disorders of the stomach).

Thirst.—*Increased thirst* is a common symptom of the most diverse disorders. It is found especially during the summer and after profuse sweating; in all febrile states; in xerostomia (dry mouth), arrest of salivary secretion; in many diseases of the stomach, especially in those attended by much vomiting; in diseases of the intestines characterized by profuse watery discharges (certain diarrhœas, cholera, hemorrhage, etc.); and in diabetes. Persistent, increased thirst should suggest diabetes and lead to appropriate examinations. The contrary condition, *absence of thirst*, sometimes more apparent than real, is found in some adynamic febrile disorders attended by stupor, especially typhoid fever. In view of its importance, large amounts of water should be given to such patients.

Flatulence.—The presence of an excessive amount of gases in the stomach may be due to several factors: (1) It may be due to the intake of air when eating (aërophagia). This is very common, and occurs especially in those who eat rapidly and bolt their food; and in nervous and hysterical subjects. The gases are largely those of the atmosphere, oxygen and nitrogen. (2) It may be due to gases taken with the food and drink—carbonated waters and wines, beers, etc. (3) It may result from fermentation of the food, which is likely to occur in the event of a deficiency or an absence of free hydrochloric acid, and when the food is retained for some time in the stomach, especially in passive congestion, chronic catarrhal gastritis, carcinoma and dilatation of the stomach, etc. In these cases one may find not only oxygen and nitrogen, but also sulphuretted hydrogen, carbon dioxide, and aromatic products of acetic, lactic, butyric, and other forms of fermentation. (4) It is a common symptom in many cases of pylorospasm, often associated with hyperchlorhydria. The condition is manifested by epigastric distress, pain, and distention; the pain may be quite severe and even colicky in nature. Relief follows belching of the gas. The *treatment* must be based upon the etiological factors; and comprises also the exclusion of fermentable foods, especially milk, buttermilk, potatoes,

rare meat, etc., and the use of antifermentatives mentioned under the different types of chronic gastritis, dilatation of the stomach, etc.

Nausea and Vomiting.—Nausea and vomiting are exceedingly common symptomatic complaints, and may be due to many causes. Usually they occur together, the nausea preceding the vomiting, but either may occur alone—the most distressing and persistent nausea with inability to vomit, and sudden vomiting without antecedent nausea.

Etiology.—Vomiting may be due to: (1) Disease of the stomach; (2) disease of the abdominal viscera; (3) disease of the nervous system; (4) intoxications; (5) reflex disturbances; and (6) it may occur as a pure neuroses.

1. The diseases of the stomach occasioning vomiting are acute and chronic catarrh, perigastric adhesions, ulcer, carcinoma, and dilatation. The vomiting of acute gastritis is often related immediately to the etiological factor, and is accompanied by epigastric pain and nausea. The vomited matter consists first of the gastric contents (which may reveal the cause of the attack), then mucus, and later bile; sometimes it is streaked with blood. Vomiting is a conspicuous feature of some cases of chronic gastric catarrh, and usually occurs in from one-half to one and one-half or two hours after eating. The vomited matter consists of partly digested food and mucus, which, being ejected, usually relieves the attendant epigastric distress and nausea. Vomiting is common in ulcer of the stomach, often occurs immediately on taking food, but may be delayed for two hours or thereabout; usually it relieves the pain which the taking of food may provoke, but it possesses no especial diagnostic significance unless accompanied by hematemesis. Vomiting is rarely missed in carcinoma of the stomach, but possesses no characteristic features, unless it is persistently of the "coffee-ground" characters. In dilatation of the stomach, vomiting is common; it occurs several hours after taking food, or at intervals of several days, is profuse, and contains stagnant and putrefying food.

2. Disease of other abdominal viscera is a common cause of vomiting often believed to be due to disease of the stomach, especially cholecystitis with or without cholelithiasis, disease of the duodenum, pericholecystic, periduodenal, and pericolonic adhesions, acute and chronic pancreatitis, appendicitis, inflammatory and obstructive disorders of the intestine, acute and chronic peritonitis, etc.

3. Diseases of the nervous system frequently give rise to vomiting, especially (*a*) meningitis (with or without hydrocephalus), brain tumor, brain abscess, and embolism, thrombosis, or hemorrhage; (*b*) errors of refraction (especially astigmatism) and of muscle balance, and Ménière's disease; (*c*) locomotor ataxia—gastric crisis; and (*d*) hysteria.

4. Intoxications are a common cause of vomiting, of which the most important are the acute infectious diseases, such as scarlet fever, yellow fever, smallpox, etc.; exogenous intoxications, such as ptomaine poisoning, acute alcoholism, sewer-gas poisoning, apomorphine, ipecac, ether, chloroform, and other drug intoxications, etc.; and autogenic intoxica-

tions, such as the toxemia of pregnancy, uremia, or cholemia (diseases of the liver), acid intoxications that accompany diabetes and the cylic vomiting of children associated with ketonuria, aberrant gout, etc.

5. Reflex vomiting is common and due to a wide variety of causes, such as disease of any of the thoracic or abdominal viscera, those already mentioned as well as renal colic, gallstone colic, intestinal colic, unpleasant odors, sights, or thoughts (in some neurotic subjects), etc.

Vomiting without nausea is found in certain disorders of the brain, such as tumor or abscess and premonitory to apoplexy, in uremia and other autogenic poisonings, in hysteria, and as a neurosis of the stomach.

The vomit consists at first of the contents of the stomach. In catarrh there is often undigested food, and there is often much mucus. The vomiting of watery fluid and mucus occurs also in certain nervous disorders. Bile in the vomit (bilious vomiting) occurs after repeated and violent vomitings, and, as a rule, has no other significance, but occasionally when appearing independently of repeated vomiting efforts it is a precursor of fecal vomiting. Fecal (stercoraceous) vomiting is significant of intestinal obstruction, but has rarely been observed in other conditions, such as gastrocolic fistula, and even in neurotic conditions. Purulent vomiting occurs in phlegmonous gastritis, or after rupture of an abscess into the stomach or œsophagus. Profuse vomiting occurs in gastric dilatation. Parasites are occasionally observed in the vomit. Blood is of frequent occurrence, and of varying significance.

Hematemesis (Gastrorrhagia; Hemorrhage from the Stomach).—**Etiology.**—Hematemesis is of common occurrence and of varying import. The general causes, alike to those that occasion hemorrhage from the intestine, are such as determine hemorrhage in other parts of the body—purpura, scurvy, malignant (or hemorrhagic) forms of certain specific infections (smallpox, yellow fever, cholera, malaria, measles, scarlatina, diphtheria, etc.), leukemia, pernicious anemia, etc.; and it is rarely a manifestation of vicarious menstruation, and other neurotic conditions, such as hysteria, vasomotor ataxia, etc. The local causes are: (*a*) Hyperemia of the gastric vessels (or of the œsophageal plexus of veins), common in advanced cirrhosis of the liver, heart disease, primary splenomegaly, etc. (*b*) Inflammation, erosion, and ulceration of the gastric mucosa, such as may be provoked by irritant poisons, etc. (*c*) Peptic or other ulceration of the stomach or duodenum, including tuberculous, syphilitic, and especially carcinomatous ulceration. (*d*) Changes in the vessel walls, such as occur in amyloid disease, nephritis, etc., may occasion hemorrhage, in some cases in consequence of the formation and rupture of small aneurysms. (*e*) Trauma, external blows or internal injuries, provoked by diverse kinds of foreign bodies (chicken bones, oyster shells, etc.). (*f*) Hematemesis may also be due to rupture of an aneurysm, or to the swallowing of blood coming from the nose, throat, œsophagus, or lungs (hemoptysis).

Symptoms.—Minor hemorrhages into the stomach doubtless frequently pass unnoticed, the blood being conveyed into the intestine with

the food. When large the blood is usually vomited, though some of it usually passes into the intestine, and, being discharged with the feces, may discolor the stools. As vomited the blood is usually dark in color, clotted, though some is fluid, is acid in reaction, and often mixed with food. When the hemorrhage occurs slowly, characteristic dark granular particles (coffee grounds) are likely to appear; when it has occurred suddenly and in large amount it is usually immediately ejected and may be quite red, though some of it is likely to be clotted. The amount of blood vomited varies within wide limits from a scarcely perceptible tinge to several quarts; and the hemorrhage may be single or repeated, either in quick succession or extend over a period of days. Attending the hemorrhage are the usual evidences of internal bleeding, faintness, weakness, pallor, dimness of sight, and often collapse. Occasionally severe and even fatal collapse has occurred without the discharge of any blood by the mouth or anus.

Diagnosis.—The many causes of hematemesis should be borne in mind, as well as the fact that some patients practise deception; in case of doubt it is wisest to see the blood before committing one's self to the belief of its having occurred. Small bleedings that readily pass unnoticed or cannot be seen by the unaided eye, may be detected by tests for occult blood in the vomit or the gastric contents after a test meal, or in the feces. Differentiating hemoptysis may sometimes cause anxiety, but in hematemesis sensations of faintness and of warmth in the epigastrium often proceed the vomiting; the blood is usually dark in color, clotted, acid in reaction, and often mixed with food; the symptoms and signs point to disease of the abdominal viscera; and blood may be discharged by the bowel; whereas in hemoptysis a sensation of tickling in the throat often precedes the hemorrhage, faintness if it occurs follows the hemorrhage, and vomiting does not occur until later; the blood is bright red, frothy, fluid, alkaline in reaction, and may be mixed with mucus or purulent expectorations; the symptoms and signs point to disease of the thoracic viscera; and the sputum, if there is any, may be blood-tinged for several days.

Treatment.—The treatment of hematemesis depends upon the cause. In general it is similar to that of hemorrhage occurring in typhoid fever (page 59).

Motor Disorders.—The motor disorders of the stomach may be irritative or depressive: on the one hand, hypermotility, peristaltic unrest, spasm, and vomiting; on the other hand, atony, insufficiency of the pylorus or of the cardia, rumination, and regurgitation.

Hypermotility or *hyperkinesis* is an increased activity of the motor functions of the stomach whereby the chyme is discharged into the intestine too soon. It may occur as a primary neurosis, but is usually associated with hyperacidity, and with some forms of achylia gastrica. The condition rarely occasions any symptoms, and the diagnosis is to be made only by attempts to remove the gastric contents.

Peristaltic unrest of the stomach is a term originally applied by Kussmaul to exaggerated peristaltic action on the stomach. It is especially common in nervous and hysterical subjects, and is often associated with like disorders of the intestine. The exaggerated peristaltic movements which often begin soon after eating, and are sometimes induced by emotional disturbances, give rise to loud rolling and gurgling sounds, which may even be heard at a distance from the patient, and occasion corresponding annoyance and discomfort. The movements are sometimes visible through a thin abdominal wall. The rapid emptying of the stomach often leads to marked hunger, and the advent of ill-prepared chyme into the intestine often provokes diarrhœa. Occasionally the direction of the movement is the reverse of the normal—antiperistalsis, and the gastric contents (rarely even the intestinal contents) may be ejected by the mouth. The condition must be differentiated from the visible peristalsis of pyloric obstruction.

Spasm of the cardia sometimes occurs in regional ulceration or carcinoma; and it may be provoked by the introduction of a stomach tube, and too hasty swallowing of food or drink, especially if too hot or too cold. Apparently as a pure neurosis it is sometimes observed in neuropathic subjects, and may be acute, recurring periodically, or chronic, developing after the ingestion of food. In air swallowers (aërophagia) who ordinarily would discharge the air, cardiospasm is not uncommon, and if associated with spasm of the pylorus, sometimes leads to considerable painful distention of the stomach (pneumatosis), which, with the subsidence of the spasm, rapidly subsides with the occurrence of the gaseous eructations.

Spasm of the pylorus is a common associate of irritative lesions of the pyloric end of the stomach (hyperacidity, hypersecretion, ulcer, etc.), and is a contributing factor in the production of pneumatosis. Whether it occurs as a primary disorder is still debated. The symptoms are pain, increased peristalsis, gastric dilatation, and vomiting. Pyloric spasm has been found also in children the subjects of persistent vomiting.

Nervous belching or *eructation* is usually noisy, and, occurring paroxysmally and sometimes persisting for hours, has been observed in neuropathic subjects (women and children). The expelled gas is usually the atmospheric air which had been previously swallowed or aspirated, and may be distinguished from the eructations of gastric disorders by the absence of unpleasant odor.

Nervous vomiting is sometimes observed in hysterical subjects, being rather a regurgitation of the food and unattended by preliminary nausea and the ordinary straining efforts. Von Leyden has described a primary periodic vomiting characterized by sudden onset, recurrence at irregular intervals, and persistence for from several hours to several days, which he regards as a neurosis. A cyclic or periodic vomiting in children has also been described, and is believed to be due to acid (auto-) intoxication.

Of interest in this connection is the vomiting of blood sometimes observed in hysterical subjects without organic disease of the stomach.

Atony of the stomach (gastroparesis, mechanical or motor insufficiency, myasthenia gastrica) sometimes results from a variety of disorders which need not be discussed here (see dilatation of the stomach), and is possibly, in unusual cases, a pure neurosis. The symptoms are those of dilatation, which soon develops.

Insufficiency or *incontinence of the cardia* may result from organic disease or from nervous disorder, and is recognized by involuntary regurgitation of food, or by rumination (merycismus)—the regurgitation and chewing of food as ruminants chew their cud. The last named, as a neurosis, is sometimes observed in neuropathic subjects—hysteria, idiocy, insanity, epilepsy, etc.

Insufficiency or *incontinence of the pylorus* may be due to organic disease or nervous disorders. The condition is rare and scarcely gives rise to destructive symptoms, but may be recognized by finding bile and intestinal contents in the stomach, and by the rapid passage into the intestine of gas or water introduced into the stomach.

Treatment.—The treatment of the motor disorders of the stomach is primarily that of any local anatomical disease with which they may be associated, but attention must also be paid to the common underlying neuropathic condition. In many cases greatest benefit attends a modified rest cure, with subsequent change of climate, if possible. A "cure" at some one of the well-known health resorts is often of much benefit on account of the habits of regularity there enforced, and the attention directed to detail. In general the diet must be regulated to suit each individual patient; the bowels must be moved once daily; and rest and exercise in proper proportion must be enjoined. Mild hydropathic measures, massage, and electricity are useful adjuvants. In cases due to irritability of the motor apparatus sedatives conjoined with the general measures mentioned are of service, especially the bromides, belladonna, and hyoscyamus, and sometimes such simple medicinal measures as Hoffmann's anodyne, etc. In conditions due to diminished irritability of the motor apparatus, the best results attend the use of the general measures mentioned, but in some cases excellent results follow the use of strychnine, but especially tincture of nux vomica, beginning with 15 minims (1 c.c.) and gradually increasing to 60 to 90 minims (4 to 6 c.c.) three times a day. One must not, however, mistake mere atony for the weakness of overuse. Iron and arsenic are valuable in many cases.

Sensory Disorders.—The sensibility of the stomach may be increased (hyperesthesia), diminished (anesthesia), or perverted (paresthesia).

Hyperesthesia of the stomach, increased sensitiveness of the sensory nerves of the stomach, is sometimes observed in hysterical and neurasthenic subjects, and is manifest by the development of epigastric distress, a sense of weight, burning pain, etc.—symptoms common to many gastric disorders, and still the gastric functions seem to be performed normally.

Sometimes extreme pain seems to be provoked by the smallest amount of food, and the patient may in consequence of a voluntary starvation emaciate considerably.

Gastralgia (gastrodynia, cardialgia), an aggravated hyperesthesia, may be due to organic disorders of the stomach, such as ulcer or carcinoma; it may be part of the gastric crisis of tabes dorsalis; and it may occur as a neurosis, independently of organic diseases. The last mentioned may occur in men or women, but is much more common in hysterical, neurasthenic, and anemic women, and is often associated with disorders of menstruation. It consists of the sudden occurrence of extremely severe pains in the epigastrium which radiate toward the back, and are likely to recur at certain intervals (in some cases at night), and are independent of the taking of food or other demonstrable cause. The attack may last from a few minutes to an hour or more, and belching, nausea, hiccough, etc., may be associated; but, as a rule, the functions of the stomach appear to be well performed: the taking of food often, indeed, affords temporary relief from the pain. Mental depression and hypochondriasis not infrequently result. Care must be taken to exclude gastric ulcer, gastric carcinoma, biliary colic, and the gastric crises of tabes.

Anorexia, or *loss of appetite*, as previously mentioned, sometimes occurs as a pure neurosis, and is sometimes associated with an absence of the sense of hunger. This nervous anorexia may occur suddenly after psychic and emotional disturbances (shock, fright, unpleasant odors, etc.), and soon passes away. A more or less chronic anorexia is observed in many neurotic subjects—in young girls who, fearing to become stout, from the habit of eating little engender the impossibility of eating much; and in certain elderly and rather rotund women in whom extreme emaciation sometimes results.

Akoria, an absence of the sense of satiety, is occasionally observed in hysterical and neurasthenic subjects, and may or may not be associated with hyperorexia.

Hyperorexia (boulimia, polyphagia), in addition to the conditions mentioned under disorders of the appetite, is sometimes seen as a pure neuroses in certain neuropathic subjects, hysteria, neurasthenia, Graves' disease, epilepsy, insanity, idiocy, etc. In consequence of the inordinate consumption of food, gastritis and gastric dilatation not infrequently result.

Parorexia (perverted appetite, pica), a craving for unusual articles for food, is seen in minor grades (capriciousness) in some young children, in chlorosis, hysteria, pregnancy, etc. In exaggerated degree, amounting almost to coprophagy, it is occasionally seen in dementia, mania, idiocy, epilepsy, etc.

Treatment.—The treatment of the sensory disorders is determined by their nature and manifestations. As in the management of motor disorders, attention must always be directed to the underlying basic disorder. In some cases of persistent gastrodynia the full rest treatment

must be carried out. Milder cases may be relieved by external applications, such as a mustard plaster, a spice poultice, or a hot-water bag, cold compresses and other hydropathic applications, and remedies such as bismuth subnitrate or subcarbonate, Hoffmann's anodyne, chloroform water, aromatic spirit of ammonia, bromides, and valerian internally. Cocaine or carbolic acid are useful in some cases. In cases associated with hyperacidity (rather common) silver nitrate and belladonna, and other remedies mentioned under hyperchlorhydria, are very useful. Morphine should be avoided, but if necessary an occasional dose of codeine may be given.

Secretory Disorders.—Although the secretion of the gastric juice is directly under the control of the nervous system, perversions of this secretion may not be ascribed to nervous influences alone. Variations in the amount and quality of the gastric juice are among the most important manifestations of functional and organic disease of the stomach of the most diverse nature; and they may occur also as a pure neurosis. The gastric juice may be increased or diminished in total quantity or in the quantity of some one or more of its constituent elements—whence we may distinguish the following:

Hyperchlorhydria (hyperacidity, superacidity), the secretion of an abnormally abundant and abnormally acid (HCl) juice during digestion, is a manifestation of certain organic diseases of the stomach, especially some cases of chronic gastritis (more particularly the cases of so-called dyspepsia occurring in young subjects), and almost all cases of gastric ulcer (in which it is quite characteristic); it is common in chlorosis; and it sometimes occurs as a pure neurosis in neuropathic subjects, in whom it may be induced by emotional disturbances, shock, etc. The ferments of the stomach are believed to be usually increased with hydrochloric acid. The hyperchlorhydria of organic disease of the stomach, notably ulcer, is likely to be more or less persistent, whereas that due to neurotic influences varies much from time to time, and is often aggravated by emotional disturbances. The symptoms are variable; hyperacidity may not produce symptoms, and *symptoms* commonly attributed to hyperacidity may occur when the hydrochloric acid is normal or even diminished. Other factors seem to be—hypersecretion, gastric atony (or other organic condition), or hypersensitiveness (hyperesthesia) of the gastric mucosa (often associated with general neurasthenia). Usually there is epigastric distress and burning (perhaps extremely severe), often associated with acid eructations, coming on in from one to three hours after taking food (at the height of digestion), and disappearing with the discharge of the chyme into the intestine or after vomiting (which is rare). The epigastric distress is often associated with distention and flatulence, commonly due to pylorospasm. The appetite is usually good and the patient appears well nourished. Constipation is the rule, but the attacks of diarrhœa are rather common, and are due to the irritation caused by the discharge into the intestine of a chyme too acid to be neutralized by the bile and the pancreatic juice. The motor function of the stomach is usually

unimpaired (empty six hours after a meal), but atony and dilatation may develop. Examination of the gastric contents after an Ewald test breakfast reveals, instead of the normal 20 to 40 hydrochloric-acid content and 30 to 50 total acidity, values as high as 60 to 100 or more hydrochloric-acid content and 80 to 130 or more total acidity. Albumin is digesed rapidly; starch slowly.

Gastrosuccorrhœa (hypersecretion; supersecretion; Reichmann's disease; gastroxynsis, Rossbach), or an increased flow of (usually highly acid) gastric juice, is a perversion of the secretory function of the stomach, whereby the gastric juice is secreted independently of the presence of food in the stomach. To the hypersecretion and hyperchlorhydria a motor insufficiency is not infrequently added. In many cases the condition is a pure neurosis, but it may also be caused by persistent irritation of the gastric mucosa—whence the term acid catarrh of the stomach, proposed by Jaworski and Gulzinski, may not be altogether inappropriate. The condition is usually found in neuropathic subjects, hysteria, neurasthenia, locomotor ataxia, etc. The increased flow may be intermittent or continuous. The intermittent form (gastroxynsis, Rossbach) is the rarest. The attacks occur at irregular intervals, and are characterized by severe, often colicky pain in the epigastrium, headache, and the vomiting of a considerable quantity of clear (or yellowish), watery, highly acid fluid. The attacks usually occur quite independently of the taking of food, last for from several hours to a day or more, and disappear rather rapidly. In the intervals the patient may be quite well, or sometimes complain of indefinite dyspeptic complaints. Continuous gastrosuccorrhœa is the more common disorder. The patient complains of epigastric distress and burning, with belching, acid eructations, and vomiting of highly acid, watery fluid, mixed or not with particles of food. In the beginning the appetite is good, but later becomes impaired; thirst is usually increased, and constipation is the rule, although attacks of diarrhœa occur. The continuation of the hypersecretion when the stomach contains no food often occasions increased distress and vomiting during the night and the early morning—which are quite characteristic. In consequence of the pyloric spasm (and consequent obstruction) induced by the highly acid gastric contents, considerable dilatation of the stomach results, and in long-standing cases, marked emaciation, even suggesting carcinoma, may ensue. The diagnosis is confirmed by washing and emptying the stomach in the evening, and, in the morning before the patient has had any food, again introducing the stomach tube and securing a clear fluid, acid in reaction and rapidly digesting albumin.

Hypochlorhydria (hyposecretion; subacidity; anacidity; hypochylia gastrica; achylia gastrica).—Inasmuch as the secretion of gastric juice, or of its hydrochloric acid content may be increased, so also may it be diminished. In the majority of cases there is both a diminution in the total amount of gastric juice as well as of the hydrochloric acid content, but the hydrochloric-acid content may be disproportionately diminished or even absent, while gastric juice is still secreted; pepsin and the curdling

ferment or their zymogens are usually present. Subacidity is a feature of most cases of gastric catarrh, and is present from time to time in many cases of nervous dyspepsia. Anacidity, even an absence of hydrochloric acid, is found in some cases of chronic gastric catarrh, and in nervous dyspepsia, and it is a noteworthy feature of almost all cases of gastric carcinoma. Total absence of the gastric juice, achylia gastrica (Einhorn), was originally considered significant of complete atrophy of the gastric mucosa, but is now known to occur as a pure neurosis, and to be susceptible of cure. The symptoms vary much in different cases. Generally they are those of chronic catarrh of the stomach with fermentation, but symptoms may be absent if the motor activity of the stomach remains normal. The condition is susceptible of accurate diagnosis only by analyzing the gastric contents after a test meal (absence of hydrochloric acid, of pepsin, and of rennin). In many cases despite the notable deficiency of gastric juice, the digestive functions seem to be well performed, a fact compatible with the demonstration recently that the intestine is able vicariously to perform the functions of the stomach when the hydrochloric-acid deficit is marked; but lienteric diarrhœa is not infrequent.

Treatment.—The treatment of the secretory disorders of the stomach is primarily that of the underlying disease—anatomical, neurotic, or both. In addition, in cases of hyperacidity, regulation of the diet is of the utmost importance. This should be largely albuminous (meat, eggs, fish, etc.), and non-irritating; starches should be given in small amounts, in view of the marked reduction in the time of amylolytic digestion in the stomach caused by the prompt and excessive secretion of hydrochloric acid; fats are fairly well tolerated, as a rule. The drinking of moderate, and sometimes of considerable, amounts of water at and between meals is often of service—serving to dilute the highly acid gastric juice; but discretion must be used in advising this, bearing in mind its influence in gastric atony and dilatation, which may be present. Alkaline mineral waters are especially serviceable. Alcohol, tea, coffee, and stimulating condiments, and too much table salt, must be avoided. The administration of alkalies, such as sodium bicarbonate, magnesia, etc., at the height of digestion, seems to neutralize the excessive acidity, but they should not be given when the stomach is empty, since thus they may only serve to increase the acidity. In many cases the hyperchlorhydria may be controlled by sedatives, of which the bromides serve as a type, but especially silver nitrate and belladonna or hyoscyamus. Cocaine and codeine may prove useful in some cases. Large doses of nux vomica are often useful in cases associated with neurasthenia.

In gastrosuccorrhœa, similar regulation of the diet is called for: the amount of water should be reduced; the meals should be reduced in number (as compared with hyperchlorhydria), but one should bear in mind that a small amount of food will sometimes almost immediately relieve the subjective complaints. Lavage is of the greatest service; one may use alkaline solutions or silver nitrate 1 to 1000. The treatment

is otherwise similar to that of hyperchlorhydria. Atropine has rendered excellent service in some cases.

In subacidity and anacidity the treatment is largely that of the underlying condition—gastric catarrh, carcinoma, etc. The diet should be carefully regulated and mixed, and should contain articles likely to excite secretion, condiments, etc. Meat is ill borne in many of these cases, and usually must be reduced to a minimum, for a time at least. Dilute hydrochloric acid and nux vomica (in large doses) are of especial value. Bitter tonics are also useful. In achylia gastrica the use of hydrochloric acid, nux vomica, bitter tonics, hydrotherapy, massage, and electricity are indicated.

NERVOUS DYSPEPSIA.

(*Nervous Dyspepsia, Leube; Gastric Neurasthenia, Ewald.*)

Nervous dyspepsia is a syndrome made up of varying combinations of the gastric neuroses, a condition, however, in which the subjective complaints are altogether disproportionate to the anomalies of gastric motility and secretion.

Etiology.—Nervous dyspepsia is ascribed to undue irritability of the gastric nerves, but in some cases the gastric condition is depressive rather than irritative. It sometimes appears as an apparently independent disorder; usually it is associated with other neurotic manifestations, such as hysteria, neurasthenia, etc.; and it may be induced in neuropathic subjects by overwork, anxiety, sexual excesses, chronic alcoholism, etc. It sometimes arises reflexly from disease of other organs, especially the eye (eye strain), and the female genitalia; it appears occasionally to follow infections, such as malaria; and it may develop in anemic and chlorotic conditions.

Symptoms.—The symptoms are extremely variable, and this is a characteristic feature. The appetite varies considerably: sometimes it is normal, sometimes increased, sometimes diminished, sometimes capricious or perverted. The tongue is usually clean. Epigastric distress is the chief complaint. Usually it is present or at least most marked during digestion; it varies in intensity from slight discomfort to extreme agony; it may come on early or not for an hour after taking food, and it may or may not be dependent upon the character of the food. The pain is most marked when there is hyperacidity. Occasionally there is epigastric tenderness; usually cutaneous hyperesthesia. Evidences of motor disturbances, such as belching, pyrosis, peristaltic unrest, etc., are not uncommon. Nausea, rarely vomiting, and referred symptoms such as headache, somnolence, palpitation of the heart, etc., may be present. When the stomach is empty there may be complaint of weakness, headache, vertigo, tinnitus aurium, etc., and sometimes even of severe pain (gastralgokenosis), which fact has led Boas to distinguish a special form of gastric neurasthenia. The motor power of the stomach

usually suffers no noteworthy deterioration, since the stomach is usually found empty six hours after a meal. Not infrequently there are evidences of neurotic disorders of the intestine—flatulence, peristaltic unrest, colic, constipation alternating with diarrhœa, etc. All of the symptoms are extremely irregular; they vary much from time to time; they develop slowly or suddenly, and disappear equally slowly or suddenly; and the attacks which may be induced by emotional or other disturbances (even ill humor) last varying periods. Equally variable is the condition of the gastric juice—subacidity, hyperacidity, and normal acidity, occurring from time to time, although independently of discoverable cause. The general health of the patient, despite the marked complaints, is often wonderfully preserved; but some aggravated cases are seen in lean and quite marantic subjects. A general hypochondriacal and neurasthenic habitus is common.

Diagnosis.—The marked variability in the complaints and in the condition of the gastric juice, the not uncommon association of other evidences of neurasthenia, the long duration of the symptoms with intervals of good health, the dependence of attacks upon well-known emotional disturbances, and the absence of specific symptoms, serve to exclude chronic gastric catarrh, gastric ulcer, and gastric carcinoma. Frequently, however, serious organic disease is manifested first by evidences of nervous dyspepsia.

Treatment.—The treatment is practically that of neurasthenia. Great difficulty is often experienced by convincing the patient that his stomach is capable of digesting a generous meal—which, when once accomplished, is soon followed by dissipation of many of the subjective complaints. Change of scene, of climate, and sometimes of association, are of much benefit in most cases. In other cases the complete or modified Weir Mitchell rest cure must be employed. Outdoor exercise, hydrotherapeutic measures, and nux vomica are very useful in other cases.

ACUTE CATARRHAL GASTRITIS.

(*Acute Gastritis; Acute Gastric Catarrh; Simple Gastritis; Acute Dyspepsia.*)

Acute catarrhal gastritis is an acute inflammation of the gastric mucous membrane, characterized by local pain and tenderness, nausea, vomiting, and sometimes fever.

Etiology.—The affection occurs at all ages, and in both sexes. An inherited predisposition is suggested by the expression "weak stomach," but the "weak stomach" is frequently acquired rather than inherited. The gouty, the debilitated, and the subjects of chronic congestion of the stomach (liver cirrhosis, heart disease) are also predisposed to the disease. The exciting cause of the disorder is summed up in the term dietetic indiscretions. These may consist of the eating of improper food, such as tainted meats, unripe fruit, etc.; of overloading the stomach with too much food and drink, even of good quality; of defective masti-

cation, often due to hurried eating, "quick luncheons," imperfect teeth, etc.; of overindulgence in alcohol, periodical debauches, etc. Acute gastric catarrh may be provoked also by the use of certain drugs, such as the iodides, salicylates, mercury, etc., and it is common also in many infective diseases, and may be aggravated by injudicious dieting.

Pathology.—The lesions are those of catarrhal inflammation in general—swelling and hyperemia, and increase of mucous secretion. Later there may be small submucous hemorrhages and superficial erosions. The cells of the mucous and peptic glands show cloudy swelling, and between them there is a small amount of round-cell infiltration. The gastric juice is deficient, and lactic and fatty acids, the consequence of decomposition, may be present.

Symptoms.—In the ordinary cure of acute indigestion the symptoms are relatively mild, and consist of epigastric distress and tenderness, soon succeeded by nausea and vomiting, with some relief. The tongue is flabby and coated with a thick, brownish-yellow fur; there is an unpleasant taste in the mouth, and perhaps headache and constipation (biliousness). In children there may be diarrhœa, and even mild intestinal colic. The attack usually subsides within twenty-four hours. In severer cases the onset may be quite acute, sometimes with a chill and moderate fever. The epigastric pain and tenderness are more marked, and the vomiting may be repeated and persistent, and bile may appear in the vomited matter. There may be acid eructations without vomiting. The appetite is lost. Thirst is usually marked, and when quenched by water is often immediately followed by vomiting. The headache is usually well marked, the tongue coated, the breath fetid, and there is much prostration, and usually constipation. The urine is concentrated and may contain indican. The attack usually subsides within three or four days, but may last longer in debilitated subjects.

Diagnosis.—Mild cases present no diagnostic difficulties. Some of the severer cases, simulating an infection at first, may be mistaken for typhoid fever when they occur independently of known dietetic indiscretions; diagnostic difficulties are sometimes maintained by persistence in the use of the term "gastric fever." From typhoid fever these cases may be distinguished by the abrupt onset, relatively marked remissions, and sudden fall of the temperature, the absence of epistaxis, of enlarged spleen, of initial bronchitis, of roseola, and of the Gruber-Widal reaction. It may simulate meningitis. In children confusion with the exanthemas may occur, but the detecting of a rash clears the diagnosis.

Prognosis.—The prognosis is good, but in debilitated subjects the disease may be unduly protracted, and as a complication of certain infectious diseases, heart diseases, etc., it may prove serious, even fatal. Recovery is usually complete, but repeated attacks may result in subacute or chronic gastritis.

Treatment.—Mild cases require no special treatment, aside from abstention from food, and a purge of castor oil, calomel, or blue mass,

followed by a saline cathartic. If there be discomfort without vomiting, vomiting may be promoted by warm water, or emetics like wine of ipecac or mustard, or lavage may be resorted too. In severe cases the patient should be confined to bed, food withdrawn for a day, and an initial dose of blue mass (5 grains, 0.3 gram), or calomel in $\frac{1}{8}$ grain (0.008 gram) every fifteen minutes for eight doses, given. The epigastric distress and tenderness, as well as acid eructations, may be relieved by bismuth subcarbonate and sodium bicarbonate, 10 grains (0.06 gram) of each, every two hours; or bismuth subnitrate (10 grains, 0.06 gram) and carbolic acid (1 minim, 0.06 c.c.) every two hours. A spice poultice to the epigastrium is often grateful. If the vomiting is severe and prolonged, it may be relieved by cocaine ($\frac{1}{6}$ grain, 0.01 gram) in lime water every hour or two, if required. Morphine had best be withheld. On the second day peptonized milk may be commenced, in small amounts, a tablespoonful (15 c.c.) every fifteen minutes. Later, the amount may be increased and the intervals lengthened, and other articles of diet added.

PHLEGMONOUS GASTRITIS.

(*Acute Suppurative Gastritis.*)

Etiology.—Phlegmonous gastritis is a rare disease, characterized by suppurative inflammation of the submucous and muscular coats of the stomach. It appears occasionally to be idiopathic, but is more common in association with pyemic processes, such as puerperal infection, etc.; it may follow trauma, and may be associated with gastric carcinoma (Osler's cases).

Pathology.—The purulent collection may be circumscribed (abscess) or diffuse (phlegmon). Rarely the abscess ruptures into the stomach cavity or the peritoneum.

Symptoms.—The local symptoms are those of severe gastritis—epigastric pain, tenderness, nausea, vomiting; the general symptoms, those of sepsis—repeated chills, high fever (104° to 105° F.), sweats, prostration, delirium, feeble pulse, leukocytosis, etc. Rarely, the pus has occasioned an epigastric prominence, and it may appear in the vomit.

The diagnosis is scarcely possible, and treatment is unavailing.

TOXIC GASTRITIS.

Etiology.—Toxic gastritis results from the ingestion (often with suicidal intent) of corrosive poisons, such as strong acids and alkalies, as well as certain non-corrosive poisons, such as phosphorus, arsenic, mercury, antimony, iodine, hydrocyanic acid, alcohol, etc.

Pathology.—In some relatively mild cases the lesions are those of a severe acute gastritis. The lesions due to non-corrosive poisons consist of fatty degeneration of the gastric tubules, with round-cell infiltration, and

foci of hemorrhage. Following the ingestion of the corrosive poisons the lesions vary, depending upon the concentration of the poison, from foci of necrosis surrounded by areas of inflammatory reaction, to the most severe and widespread destruction of the stomach wall, amounting in some cases to conversion of a large portion of the stomach into a discolored eschar, and perforation into the peritoneum. Similar lesions are usually found also in the mouth, pharynx, and œsophagus, and rarely in the duodenum. Should the patient survive, ulceration is followed by cicatrization and atrophy of the gastric mucosa; sometimes by hour-glass contraction of the stomach, stenosis of the œsophagus, etc.

Symptoms.—In severe cases the ingestion of the poison is followed by extreme abdominal pain, immediate collapse, and speedy death. In less severe cases the symptoms consist of severe burning pain in the mouth, œsophagus, and stomach; swallowing is almost, if not quite, impossible, and vomiting and retching almost constant. The vomit is bloody, and sometimes contains sloughs of the stomach wall. The abdomen is tender and tympanitic; the urine may contain albumin and casts. Subcutaneous hemorrhage and delirium and convulsions may develop, and the patient may die in a few days from the intoxication, or later, from perforation of the stomach or inanition. In mild cases recovery may ensue.

Diagnosis.—The diagnosis is quite apparent from the history of the case, and an inspection of the mouth, throat, and vomit.

Treatment.—The treatment is that appropriate in the different poisonings to which the condition may be due, especially the use of magnesia with milk or egg-albumen in acid poisonings, and dilute acids in strong alkali poisonings, etc. Lavage may prove useful. Morphine should be given for the pain. In severe cases treatment is usually unavailing.

INFECTIOUS GASTRITIS.

The term infectious gastritis is sometimes employed to distinguish affections of the stomach due to certain microörganisms, and occurring usually in the course of certain infective diseases. In a broad sense, some of the cases of acute catarrhal gastritis are doubtless infective; phlegmonous gastritis is, of course, infective. Usage, however, has tended to restrict the term to diseases of the stomach caused by microörganisms, such as the diphtheria bacillus, tubercle bacillus, pneumococcus, the typhoid bacillus, colon bacillus, streptococcus, etc. Occasionally, especially in diphtheria, pneumonia, pyemia, typhus fever, smallpox, etc., the lesions go on to the formation of a pseudomembrane—sometimes called membranous gastritis. Rarely fungi, such as yeast fungi, Saccharomyces albicans, the favus fungus, etc., excite a gastritis—so-called mycotic or parasitic gastritis. The symptoms are not distinctive, and the nature of the lesions is not to be recognized unless, perchance, the fungus or a portion of the pseudomembrane is detected in the vomit.

CHRONIC GASTRITIS.

(*Chronic Catarrhal Gastritis; Chronic Gastric Catarrh; Chronic Dyspepsia.*)

Chronic gastritis is a chronic inflammation of the gastric mucosa attended by increased secretion of mucus and alterations in the gastric juice, and sometimes associated with impairment of the motor power, and different structural changes in the wall of the stomach.

Etiology.—Chronic gastritis may follow directly on an acute attack, but it is more commonly due to the continuous or frequently repeated action of minor irritants, especially in those predisposed by constitutional or local factors. The general predisposing factors comprise anemia (chlorosis, pernicious anemia) and the toxic conditions and vitiation of the blood, inseparable from tuberculosis, gout, nephritis, diabetes, etc. The local predisposing factors comprise the congestive conditions found in cirrhosis of the liver, chronic heart disease, etc., and different diseases of the stomach itself, such as carcinoma, ulceration, dilatation, etc. The exciting cause of the condition comprises dietetic indiscretions in a broad sense, such as persistent hurried and irregular eating, insufficient mastication, too highly seasoned food, pastry, overindulgence in alcohol, tea, tobacco, iced drinks, soda water, etc. It is sometimes induced also by the long-continued use of certain drugs, such as arsenic, mercury, iodine, and drastic cathartics.

Pathology.—The lesions vary considerably in different cases. In cases, mild and of short standing, so-called simple chronic gastritis, the mucous membrane is thickened, pale gray in color, somewhat congested, though less so than in acute gastritis, and covered by a tenacious mucus, which sometimes is so worked as to warrant the term mucous gastritis. Many dilated and enlarged veins may be visible, especially in the gastritis of portal obstruction, and there are frequently small areas of hemorrhage and pigmentation—occasionally of superficial erosion. The glands are usually choked with retained secretion and may enlarge to form small cysts. In more aggravated cases, almost, if not quite, the entire wall of the stomach participates in the process, and the mucous membrane may be thrown up into small polyp-like projections—gastritis polyposa. Histologically the lesions are those of interstitial inflammation with parenchymatous degeneration. In most cases, after the lapse of time, atrophic alterations, and more or less dilatation of the stomach, result—so-called atrophic gastritis. The mucous membrane, as well as the entire wall of the stomach, becomes much thinned, the gastric tubules atrophy and may eventually disappear and be replaced by sclerotic fibrous connective tissue, giving to the mucous membrane an exceedingly smooth appearance, which may be interrupted here and there by a small cyst. In rare cases the stomach becomes much diminished in size (so that its capacity may be only a few ounces), and its wall very much thickened (2 to 3 cm.) in consequence of hyperplasia of the submucosa and muscularis (sclerosis of the stomach). The condition may resemble a

diffuse infiltrating carcinoma. This and the atrophic gastritis are sometimes spoken of as two varieties of sclerotic gastritis—the one as phthisis ventriculi, the other as cirrhosis (sclerosis) ventriculi.

Symptoms.—The symptoms vary much in different cases, and in the same case from time to time. Usually the appetite is poor and eating is followed by epigastric distress and a sense of weight and fulness which sometimes amounts to severe pain, and the epigastrium may be tender on pressure. In some cases the appetite is good; in other cases there is marked distress when the stomach is empty, and eating only augments it. Acid eructations and flatulence are frequently present (flatulent dyspepsia). Nausea is common, but vomiting is rather unusual except in aggravated cases, in alcoholic subjects (the morning vomiting of alcoholics), and in the event of serious gastric disease, such as carcinoma, ulcer, pyloric obstruction, dilatation, etc. Associated with the gastritis there is usually more or less catarrh of the mouth and the intestine: the tongue is coated and its tip and margin red; there is an unpleasant taste in the mouth, the breath is malodorous, the oral and pharyngeal secretions are often increased, and constipation (due to deficient activity of the intestinal muscularis) is the rule, although diarrhœa, sometimes associated with tympanites and colic (due to the propulsion into the intestine of undigested and fermenting food) may occur. Headache, irritability, languor, and vertigo, manifestations of auto-intoxication, and palpitation of the heart, due also to pressure on the heart by the distended stomach, are common concomitants.

In the event of vomiting, the ejected matter consists of partly digested and sometimes fermenting food, considerable mucus, and different aromatic acids (butyric, lactic, and acetic), which partly or completely replace the normal hydrochloric acid. The presence of considerable mucus suggests the term mucous gastritis. Analysis of a test breakfast reveals variations in the hydrochloric acid—hyperacidity (uncommon), subacidity (common), or anacidity (rare). Absorption from the stomach is slow—potassium iodide given in a capsule does not appear in the saliva until considerably after fifteen minutes, the average time in normal individuals; and the motility of the stomach is impaired—food may be found in the stomach as much as seven hours after the last eating.

Atrophic or sclerotic gastritis gives rise to no characteristic symptoms. The symptoms are severe, as a rule, and the patient may emaciate rapidly. Pernicious anemia or carcinoma is frequently simulated, particularly as, in some cases, the small, thickened, and sclerotic stomach gives rise to a palpable mass in the epigastrium, and hydrochloric acid may be entirely absent from the gastric contents.

Diagnosis.—The diagnosis is usually quite apparent; but it is important to bear in mind that the great majority of cases of supposed chronic dyspepsia or gastritis that are not improved by several months of well-directed therapy have an anatomical basis. Most of them are cases of gastric or duodenal ulcer, gastric carcinoma, cholecystitis with or without gallstone, perigastric, pericholecystic, or periduodenal adhesions, chronic

pancreatitis, chronic appendicitis, cirrhosis of the liver, etc. In protracted cases it may be difficult to exclude carcinoma, especia ly as carcinoma may be preceded for years by chronic gastritis or ulcer. Rapid course, marked emaciation, hematemesis, severe epigastric pain, a palpable tumor, glandular enlargement, absence of hydrochloric acid, and the presence of lactic acid (after a Boas test meal), and the Oppler-Boas bacillus, characterize carcinoma. When a palpable tumor is present the rare cirrhotic stomach may be distinguished from carcinoma by the fact that in the one the stomach is small, whereas in carcinoma it is usually considerably dilated.

Prognosis.—In many cases the outlook is good—with regulation of the diet and proper hygiene. Atrophy of the mucosa, however, since it cannot be repaired, may lead to grave anemia and death.

Treatment.—The special etiological factors in each case must be corrected. Food must be taken at regular intervals; it must be eaten slowly and thoroughly masticated; tea, coffee, alcohol, tobacco, pastry, hot bread, hot cakes, fats, sweets, pork, veal, etc., must be prohibited. In some cases mere regulation of the diet suffices to dissipate all the symptoms. In other cases a strict dietetic regimen is imperative, for a time at least; in some cases, especially those occurring in liver cirrhosis, Bright's disease, etc., a milk diet for several weeks may be tried. The milk should be given in stated amounts at regular intervals and about 2 liters in the twenty-four hours. It may be diluted with Vichy or lime water, or, especially in the event of nausea, sodium bicarbonate (5 to 10 grains 0.3 to 0.6 gram) may be added to each glassful. For a short time the milk may be peptonized, or buttermilk may be substituted. In most cases it is unnecessary to restrict the diet to milk; most patients can digest, in addition to milk, dry toast, chicken, and roasted or broiled red meats. Farinaceous foods, especially potatoes, and coarse vegetables are a fruitful source of trouble and must be interdicted, especially in the event of flatulence or acid eructations. As the patient improves, the diet may be made more liberal by the addition of eggs, broths, and rice; and green vegetables, such as spinach, lettuce, fruits, etc., may be allowed; but pastry, fried foods, coarse vegetables, strong condiments, and meats, such as pork and veal, must be prohibited. In general, fluids taken with a meal should be moderate in amount; one should strive for the happy medium between the senselessness of the absolutely dry diet and the perniciousness of the excessive amounts of fluid. Plain water is the best fluid. It should be partaken of freely between meals.

The medicinal treatment should be based upon the state of the gastric secretion. In conditions of subacidity, dilute hydrochloric acid (15 minims, 1 c.c.), in a glassful of water, should be given with each meal. In many cases, however, instead of supplying the deficient hydrochloric acid, we can stimulate the secretion and improve the motor power of the stomach with tincture of nux vomica, beginning with 15 minims (1 c.c.) three times a day and gradually increasing the dose to 60 minims (4 c.c.) or more. The bitter tonics, gentian, cinchona, quassia, calumba,

condurango, cardamoms, etc., are extensively used, and are of value in improving the appetite and stimulating the secretion and motor power of the stomach. Digestive ferments (pepsin, pancreatic preparations, etc.) may be of service in supplying a deficient digestive ferment pending the restoration of the gastric functions, but they are less important than was formerly believed, the hydrochloric acid being the more important factor and activating the zymogens already present. In protracted cases silver nitrate, $\frac{1}{8}$ grain (0.008 gram) combined with extract of belladonna or hyoscyamus, $\frac{1}{8}$ grain (0.008 gram), three times daily, if there is much pain and hyperacidity, is of value.

In many cases lavage, preferably in the morning before breakfast, is serviceable. In ordinary cases, plain lukewarm water, or 1 per cent. sodium chloride solution, may be used. In case there is much mucus, a 5 per cent. sodium bicarbonate solution may be used, and in case of fermentation, a 3 per cent. boric-acid solution or a 2 per cent. salicylic-acid solution, may be used—depending upon the exigencies of the case. The lavage may be done daily or every other day, but its use should not be persisted in too long, lest the "stomach-tube habit" be acquired. In some cases the drinking of a glassful of hot water before breakfast, or before each meal, is a more or less satisfactory substitute for lavage.

Excessive flatulence may be relieved by the use of bismuth, sodium bicarbonate, soda-mint, aromatic spirit of ammonia, chloroform water, magnesium oxide, carbonate, or salicylate, carbolic acid, creosote, resorcin, charcoal, etc. Vomiting, if marked, may be relieved by carbolic acid, dilute hydrocyanic acid, cerium oxalate, bismuth subnitrate or subcarbonate, cocaine, lavage, etc.

ACUTE DILATATION OF THE STOMACH.

Acute dilatation of the stomach results usually from the rapid overfilling of the stomach with excessive amounts of food and drink (especially effervescing fluids)—the development of the condition being somewhat favored by acute and chronic catarrh of the stomach. Aggravated cases merit the designation acute paralytic distention of the stomach (Fagge), and not infrequently terminate fatally. Another and large series of cases follow operations and general anesthetization. Minor grades sometimes occur during or after certain of the infectious diseases —in consequence of parenchymatous degeneration of the gastric musculature. A majority of all cases have been found to be due to obstructions of the lower end of the duodenum by the root of the mesentery. The *symptoms* are sudden in onset, and consist of abdominal pain and distention, the vomiting of a large amount of bile-stained non-fecal fluid, collapse, and frequently death. The *treatment* of the aggravated cases consists of emptying the stomach by means of the stomach tube, placing the patient in the anterior prone or knee-chest position, subsequent restriction of the diet, and the use of tonics.

CHRONIC DILITATION OF THE STOMACH.

(*Gastrectasis.*)

Etiology.—Chronic dilatation of the stomach—an increase in the capacity of the stomach associated with motor insufficiency—is especially common in adult life, but is not rare in rachitic children. It may be obstructive or non-obstructive. The causes of obstructive dilatation are: (1) Pyloric obstruction, which may be due to (*a*) intragastric factors, such as cicatrization of a gastric ulcer, carcinoma or other tumor, non-malignant hypertrophic stenosis of the pylorus, pyloric spasm; or (*b*) extragastric factors, such as compression by a tumor of the gall-bladder, liver, pancreas, or neighboring lymph nodes, or adhesions about the pylorus, and other causes (such as displaced right kidney) exerting traction and causing twisting or angulation of the pylorus. Similar factors operating on the duodenum may also cause dilatation of the stomach. Non-obstructive dilatation is due to atony of the muscular coat of the stomach; this may be (*a*) congenital, or (*b*) acquired, in consequence of repeated overfilling of the stomach (beer drinkers, gluttons, the insane, diabetics, etc.), chronic catarrh, and other organic (especially degenerative) diseases of the stomach. It is common in tuberculosis, carcinoma, anemia, and other general disturbances of nutrition, disorders of innervation, etc.

Pathology.—The capacity of the average normal adult stomach is about 1600 c.c. (Ewald), though this is subject to individual variations, and doubtless varies in the same individual from time to time. Some persons normally have an abnormally large stomach—megastria. In disease, however, the dilatation may be so extreme that the stomach occupies almost the entire abdominal cavity and attains a capacity of 10 or more liters, especially in the obstructive forms. In obstructive dilatation the stomach wall is thickened in consequence of hypertrophy; in the non-obstructive form it is usually thinned in consequence of atrophy of the muscular coat. There are also the evidences of catarrhal or other alterations.

Symptoms.—The symptoms vary with the cause and the degree of the dilatation. Minor grades, especially those due to muscular atony, may be symptomless. The symptoms of chronic gastric catarrh are present in the majority of cases, and should the dilatation be associated with carcinoma, the specific symptoms of carcinoma may be present. The dilatation itself is suggested by loss of appetite alternating with excessive appetite and thirst, acid eructations, constipation, and the vomiting at long intervals of large amounts of fluid and partially digested and stagnant food. Vomiting sometimes occurs daily, but not infrequently at longer intervals—three days or more; and the amount ejected at once may be as much as 3 liters or more. The epigastric distress, which often has been gradually increasing, is usually much relieved by the vomiting. The vomit is malodorous and acid (due to aromatic acids, lactic, acetic, and butyric), and on standing separates into three

layers — the lowermost consisting of decomposing food remnants (food that may have been taken days, even weeks, previously); the middle, of a dirty, yellowish, light brownish, or grayish fluid; and the uppermost, of a dirty brown frothy mass. The hydrochloric acid content varies much, depending largely upon the associated conditions; it is usually diminished or absent, rarely increased. In consequence of the absorption of but little fluid, constipation, lessening of the amount of urine, and a dry skin result; in consequence of deficient nourishment the patient loses weight and strength and becomes anemic; and in consequence of auto-intoxication, the absorption of fermentation products, divers nervous disorders, such as headache, vertigo, insomnia, neuralgiform pains, mental depression, hypochondriasis, etc., result.

Physical Signs.—Physical examination often reveals a prominent and distended abdomen (often most marked about or below the umbilicus), and the outline of the enlarged stomach can often be perceived—the greater curvature usually below the umbilicus, often near the pubes. Peristaltic (occasionally antiperistaltic) waves are sometimes visible—and may even be palpable. Splashing sounds (clapotage) may usually be elicited, and sometimes even by the patient (by suddenly contracting the diaphragm) or other sudden motion. Percussion and auscultatory percussion yield definite information as to the size of the stomach. The most trustworthy results are obtained by inflating the empty stomach (by means of a stomach tube and a hand-bulb syringe or other pump). A stomach may be considered dilated when the vertical diameter of gastric tympany is more than 12 to 14 cm. The lower border of the stomach when it is dilated is, at or below the umbilicus, but one must not mistake a displaced for a dilated stomach. Frequently, however, the two conditions are associated.

Diagnosis.—The diagnosis is usually quite obvious from the recurrent vomiting of large amounts of fluid and the results of inspection and of percussion, especially after inflation of the stomach. Gastroptosis may usually be excluded by the results of physical examination and the presence of vomiting of stagnant food.

Prognosis.—The prognosis is favorable in most cases except those due to malignant obstruction of the pylorus.

Treatment.—The most particular attention must be paid to the diet, which should be concentrated and readily assimilated, such as eggs, chops, steak, roast beef, chicken, lamb, and mutton (no coarse meats), well-toasted bread or Zwieback, rice, etc., for a time, at least. Fluids should be reduced to a minimum, and the food should be taken in small amounts at rather frequent intervals, rather than larger amounts less frequently. The digestive processes may be aided and the comfort of the patient promoted by the recumbent posture for an hour or so after partaking of food. Gradually a more liberal diet may be permitted. In aggravated cases it may be necessary to put the patient to bed and give only small amounts of egg-albumen, peptonized milk, scraped beef, etc., for several days or a week.

Lavage is a useful adjuvant in many cases, and is of unquestionable service in the beginning of the treatment. Warm water, a saline solution, or a mild antiseptic solution (boric acid, salicylic acid) may be used. It serves to remove the stagnating and fermenting food, to cleanse the mucous membrane (removing sometimes large amounts of mucus—in associated catarrh), and it relieves the overloaded stomach of a great weight. It is wise to discontinue the lavage as soon as possible, since the stomach-tube habit is not without attendant evil consequences.

Medicinal measures comprise the use of tincture of nux vomica, which is of the very greatest service in increasing the motor power of the stomach, if given in doses of 15 minims (1 c.c.), three times daily, and gradually increased until as much as 60 minims (4 c.c.) or more is given three times daily. Strychnine may be used, but it appears to be not so useful as the tincture of nux vomica. When hydrochloric acid is deficient, it should be supplied for a time at least. Occasionally antifermentatives, such as carbolic acid, creosote, salicylic acid, resorcin, charcoal, magnesia, etc., may be called for.

In dilatation due to benign or malignant obstruction of the pylorus, perigastric adhesions, and similar conditions, resort should be had to surgical procedures.

ULCER OF THE STOMACH AND DUODENUM.

(*Simple, Round, Peptic, or Perforating Ulcer of the Stomach and Duodenum.*)

The term ulcer of the stomach or duodenum is somewhat inaptly used to designate a localized area of necrosis of the wall of the stomach or duodenum, resulting from lessening of vitality and consequent digestion by the gastric juice.

Etiology.—Gastric and duodenal ulcer may occur in both sexes, and at all ages, even in the very young and the aged. Formerly about 60 per cent. of the cases were believed to occur in women, but surgical statistics have shown that the condition is just as common if not more common in men. In women it occurs especially between the twentieth and the thirtieth year, particularly in servants, in whom it is attributed to anemia and their injudicious mode of eating and tasting very hot food (of the truth of which there is little trustworthy data). In men it is most common between the thirtieth and the fiftieth year, especially in shoemakers, tailors, and weavers, etc., in whom, with little reason, it has been attributed in part to pressure on the epigastrium.

The exact cause of the necrosis is ill understood. It has been attributed to trauma — external, internal, and that resulting from violent vomiting (whence, as sometimes in uremia, hemorrhagic erosion and ulceration may result); by Virchow to thrombosis or embolism of a nutrient artery, infarction, and consequent necrosis; by Pavy and Cohnheim to diminished alkalescence of the blood (common in anemias), supplemented by gastric hyperacidity and local injury to the gastric

mucosa; by Dallavedova to disturbances of the celiac plexus and the splanchnics; by Talma to disease of the pneumogastric nerve, occasioning undue muscular contraction; and by Letulle, Nauwerck, and Sidney Martin to bacterial infection and toxic necrosis of the gastric mucosa. Recently it has been suggested that in health the cells of the gastric mucosa produce an anti-enzyme that resists peptic digestion of the cells, and that this anti-enzyme may be deficient or absent in disease. And it has also been supposed that the system may contain a poison which, being eliminated, injures the gastric cells. Experimentally, ulcers may be produced by injecting into the stomach a gastrotoxic serum prepared by immunizing a goat, cat, or other animal to the gastric cells of another animal Ewald insists upon the importance of anemia and gastric hyperacidity, which are assuredly present clinically in most of the cases.

The analogous duodenal ulcer is attributable to like factors. In addition, ulceration of the duodenum is often observed after burns of the skin, especially of the abdomen; it is attributable to the action of toxins formed in the burned skin (or, perhaps, retained on account of functional incapacity of the burned skin), and eliminated by the duodenal mucosa.

Peptic ulceration of the jejunum is occasionally observed after gastroenterostomy, and is attributed to the influence of acid chyme, which finds its way directly into the intestine.

Pathology.—Small abrasions or hemorrhagic erosions of the gastric mucosa are rather common. They are perhaps not essentially different from the more obvious ulceration (so-called), although they usually heal. The gastric ulcer itself may be acute or chronic. Usually (80 per cent. of cases) there is a single ulcer (chronic form); sometimes there are several, and rarely there are many ulcers (acute and chronic forms): I have seen more than twenty in one case. The single chronic ulcer is usually situated in the posterior wall toward the pylorus and at or near the lesser curvature (more than 80 per cent. of the cases); in some cases it involves and may even straddle the pylorus. The acute ulcer is usually small, rounded, well defined (punched out), with a smooth floor, and without associated induration. The chronic ulcer is often much larger, sometimes very large, 5, 10, or 15 cm. in diameter, irregular in shape, with sloping or terraced margins (funnel-like); its floor may be the submucous, muscular, or peritoneal coats of the stomach—in some cases even adjacent viscera, such as the pancreas or liver, to which it may have become adherent. Cicatrization usually results, the resulting scar being smooth if the mucous membrane only is involved; but the cicatrization of deeper ulcers is attended by the development of a puckered and stellate scar and consequent damage to the stomach, such as pyloric stenosis and dilatation of the stomach. The cicatrization of a so-called girdle ulcer may lead to hour-glass contraction of the stomach; and perigastric adhesions may result in other deformities of the stomach, interference with its function, and persistent pain. Perigastric, especially posterior, adhesions, however, in many

cases are a fortunate provision of nature, since they anticipate, and sometimes prevent, a not uncommon and frequently fatal accident—perforation. Perforation, when it occurs, is usually into the adhesions and the related organs, where it gives rise to an abscess, or into the lesser peritoneal cavity, where it occasions what is known as a subphrenic pyopneumothorax; or, as sometimes happens, sinuses may form between the stomach and the colon, duodenum, pericardium, pleura, or externally. Perforation of ulcers on the anterior surface of the stomach, where adhesions rarely form, usually gives rise to an extensive and rapidly fatal peritonitis. Extensive and rarely fatal hemorrhage may occur usually from a branch or the main artery of the lesser curvature, sometimes from rupture of an aneurysmal dilatation of an artery in the floor of the ulcer, rarely from erosion of the splenic artery, etc. More common bleeding is doubtless prevented by antecedent thrombosis. Some ulcers persist for years; recurrences are common; and carcinoma sometimes develops from an ulcer or its cicatrix.

The analogous duodenal ulcer is found above the papilla. In more than one-half of the cases it reaches to within three-fourths inch of the pylorus, and in 20 per cent. of the cases it involves the margin of the pylorus. The appearances and the accidents, especially hemorrhage, perforation, and adhesions to adjacent tissues are quite analogous to those of gastric ulcer.

Symptoms.—The symptoms are extremely variable. In a few cases (latent gastric ulcer) there are no symptoms, the ulcer being an accidental finding at the necropsy; rarely a profuse hemorrhage or a perforation is the initial manifestation. In the majority of cases, however, definite symptoms suggesting ulcer are preceded for months or years by the ordinary evidences of chronic gastritis: but many of these are cases of already existing, that is, chronic ulcer. Ulcer is suggested by pain, tenderness, undue vomiting, pylorospasm, and hyperchlorhydria. Gaseous and acid fluid eructations, headache, faintness, and constipation, occasionally diarrhœa, may also be present. Pain is a constant and characteristic symptom, although it varies much in different cases. It is usually severe, and may be extreme and paroxysmal (characteristic). It may be localized to a small spot, which is often exquisitely tender, near the median line in the epigastrium; it often radiates to the side and back, and may be associated with a spot of tenderness near the tenth thoracic vertebra (Boas' point). Taking food often induces or aggravates the pain, either immediately, or after a short time, and the pain persists until the stomach is relieved of food, either by vomiting or by its passage into the intestine; it is sometimes very severe two or three hours after eating, when the acid chyme begins to pass into the intestine, thus irritating an ulcer situated at the pylorus. The pain is sometimes relieved by firm pressure or by change of posture—permitting the gastric contents to gravitate away from the site of the ulcer. The pain is thus due to direct irritation of the ulcer by the gastric contents, to the peristaltic movements of the stomach, and to the hyperchlorhydria

(usually, though not always, present). In some cases it may also result from an intermittent or more or less continuous pylorospasm, which leading of retention of intragastric gases may cause stretching of the stomach or increased intragastric tension.

Vomiting is rather common, and occurs usually at the height of the paroxysm of pain (from one-quarter to two hours after eating). It is not infrequently provoked voluntarily by the patient to relieve the intense pain. When perigastric adhesions have formed, vomiting may occur in the intervals between eating, and when dilatation (a late phenomenon) ensues, the characteristic vomiting of this condition results. In the absence of dilatation, gastric digestion is usually hastened rather than retarded, and the vomited matter is usually small in amount, well digested, and has an acid taste and odor. It also, as well as a test meal, usually but not invariably reveals marked hyperacidity (increased hydrochloric acid).

Hematemesis is common—occurring clinically in about 50 per cent. of the cases. Some bleeding probably occurs in all cases, from irritation and congestion provoked by the food, distention and stretching of the stomach by gases (Moynihan), severe vomiting, etc., but the blood may not be visible macroscopically in the vomit or the stools. As a rule, the bleeding is moderate in amount, but it may be extreme (and followed by syncope), and it is rarely immediately fatal. The extravasation of the blood being, as a rule, rather slow, the blood may remain in the stomach for some time, and, being altered by the gastric juice, appear in the vomit as black, tarry masses resembling liver, or smaller particles resembling coffee grounds. The vomiting of a large amount of bright blood, however, is quite significant of gastric ulcer. Altered blood often appears in the stools; and in a few cases, when the hemorrhage is small, it may appear in the stools and not in the vomit. In many cases recourse must be had to tests for occult blood to detect small amounts in the vomit or the feces. In most cases a posthemorrhagic anemia develops, and in some cases may become extreme.

In some cases one may be able to palpate, through the abdominal wall, a mass, which may be a thickening about the ulcer, perigastric exudation and adhesions, enlarged regional lymph nodes, or the hypertrophied pylorus resulting from long-continued spasmodic contractions. Loss of weight may occur in protracted cases and following large hemorrhages (when it is associated with marked anemia), but many subjects appear fairly well nourished.

The onset of perforation, which occurs in about 6 per cent. of the cases, is manifested by severe abdominal, usually epigastric, pain, and shock, and the rapid development of peritonitis, usually of the upper half of the abdomen, although it may become general.

Diagnosis.—Dyspeptic symptoms, with local pain occurring paroxysmally and provoked by food, circumscribed tenderness, hyperchlorhydria, and hematemesis in a young woman, are unmistakable; but dyspeptic symptoms, without hematemesis, in a chlorotic girl or in a man may be difficult to interpret. The greatest diagnostic significance

should be attached to the persistence of symptoms, especially of pain, tenderness, pylorospasm, and hyperchlorhydria, despite several months of well-directed therapy: such cases often believed to be merely "chronic indigestion," usually have an anatomical basis, which is often ulcer. The pain of ulcer, as contrasted with other conditions, may sometimes be relieved by orthoform, 10 to 30 grains (0.6 to 2 grams) at once, or smaller doses before meals. Some diagnostic significance has been attached to this. It may be difficult or impossible (and is usually unnecessary since the treatment is similar) to differentiate duodenal from gastric ulcer, but duodenal ulcer is suggested by the male sex, later life, pain to the right rather than to the left of the median line, delayed after taking food, little or no vomiting, and blood in the stools rather than hematemesis. In gastralgia, as contrasted with ulcer, the pain is less circumscribed, occurs independently of or is relieved by food (this occasionally occurs in ulcer), there is less circumscribed epigastric and post-thoracic tenderness, there is no hematemesis, vomiting is uncommon, the general nutrition of the patient suffers little if at all, and there are periods of entire relief from symptoms. The pain of biliary colic may be distinguished by its sudden onset, usually extreme severity, short duration, and sudden cessation, enlargement and tenderness of the liver and gall-bladder, and possible jaundice, fever, and leukocytosis. The hematemesis of cirrhosis of the liver may be distinguished by its marked preference for the male sex, certain etiological factors (especially alcohol), and associated liver conditions. The hematemesis of chronic gastric catarrh, especially that occurring in the aged, and that attributed to gastric erosions, is usually difficult of differentiation, but in erosions hematemesis is less marked, the pain is less severe and burning in character, subacidity rather than hyperacidity is likely to be present, and, as maintained by Einhorn, shreds of the gastric mucosa may be found in the vomit or the washings after lavage. Attempts have been made to determine the site of the ulcer by noting the influence of posture on the pain (different postures causing the food to come in contact with and irritate the ulcer, or to gravitate away from it); and a palpable induration has been deemed significant of ulcer on the anterior wall, and pain in the back significant of ulcer of the posterior wall—but these signs are untrustworthy.

Prognosis.—Most patients recover, but the disease usually runs a protracted course, relapses are common, and large hemorrhages and perforations are to be dreaded; cicatrization of an ulcer may cause pyloric obstruction and dilatation or hour-glass contracture of the stomach, or may be followed by carcinoma; and perigastric adhesions may be a source of continued pain and ill health.

Treatment.—Absolute rest in bed for the patient for at least four weeks, and rest for the stomach secured by rectal feeding for at least seven days, are of prime importance. Rarely it may be deemed desirable to begin feeding by the stomach at the end of three or four days. The rectal enemas may consist of peptonized milk, 4 ounces

(120 c.c.), the whites of one or two eggs, a pinch of salt, and 5 minims (0.3 c.c.) of deodorized tincture of opium. This may be injected high up in the rectum or in the sigmoid every six or eight hours, and should be preceded by a cleansing enema. When feeding by the mouth is commenced the food must be very bland, and neither too hot nor too cold. Milk is the best food. At first it should be peptonized or pancreatinized, and given in small amounts at regular intervals—in ordinary cases, 2 to 4 ounces, and later 4 to 6 ounces, every three hours. To counteract the hyperacidity, sodium bicarbonate or calcined magnesia may be added to the milk. Buttermilk is sometimes an excellent substitute. Soon two to four or six raw eggs daily may be added to the milk, and beef juice, thin gruels, arrowroot, strained broths, potato puree, etc., may be given. In the early days of the treatment undue thirst may be relieved by enteroclysis. At the end of three or four weeks from the time of beginning feeding by the mouth, scraped beef, sweetbread, tender chicken, and similar articles of diet may be allowed; but care and discretion must be enjoined for at least three months.

An essential feature of the treatment is regulation of the bowels, which should be opened once daily—preferably by the use of saline cathartics. These do good not only by clearing the bowel, but also (and this is the philosophy of their use in large amounts) by counteracting the hyperacidity, and by their depurative action relieving the commonly associated gastro-intestinal catarrh.

In many cases, aside from the foregoing, no medicines are required—unless pain is severe. Leube speaks highly of the local use (to the epigastrium) of boric-acid ointment and hot flaxseed poultices (changed every fifteen minutes) during the day, and a cold-water compress during the night—in relieving the epigastric pain and distress. Hemorrhage (blood in the bowels, as well as hematemesis) is a contra-indication to the use of the poultices. In cases of severe pain, however, I am rather partial to the use for a short time of opium, which should be combined with silver nitrate, giving three times daily a pill of silver nitrate, $\frac{1}{4}$ grain (0.015 gram), and extract of opium, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008 to 0.015 gram). If the pain is not severe, I substitute hyoscyamus or belladonna for the opium. The bromides also are useful in relieving the pain and in checking hyperacidity. Codeine or cocaine may be used for the same purpose; and these, as well as cerium oxalate, hydrocyanic acid, chloroform, ether, etc., are useful in protracted vomiting. Bismuth in large doses (30 to 60 grains, 2 to 4 grams, three times daily) has been highly recommended, but it probably exerts more beneficial influence on the associated catarrh than on the ulcer, and it often gives rise to the most annoying constipation. Olive oil in large doses has been recommended by Cohnheim.

In the event of severe hemorrhage the patient must be confined to bed absolutely at rest; an ice bag may be applied lightly to the epigastrium, and morphine, $\frac{1}{4}$ grain (0.015 gram), should be administered hypodermically. Tannic acid, gallic acid, lead acetate, ergot, Monsel's

solution of iron sulphate, are of doubtful utility; but 30 minims (2 c.c.) of adrenalin chloride (1 to 1000) may be administered with the hope that it may do good. Hypodermoclysis should be resorted to in large hemorrhages to restore the volume of fluid lost.

The surgical treatment of gastric ulcer has to be considered in many cases. Operation may be said to be indicated: (1) In recurring hemorrhages uncontrolled by medicinal measures; (2) in intractable cases with severe pain, persistent vomiting, hematemesis, and resulting emaciation; (3) when perforation has occurred; and (4) for the relief of perigastric adhesions, and the less common perigastric, subphrenic, or other peritoneal suppurations.

CARCINOMA OF THE STOMACH.

Etiology.—Carcinoma of the stomach is somewhat more common in males than in females; and occurs especially after the fortieth year (80 per cent. of the cases); but it has been observed in subjects less than twenty-five years of age, and rarely even in children. The etiological factors are the same as those of carcinoma in general, and are as ill understood. Some influence is attributed to heredity. The affection is more common in the white race than in the negro, and in some countries (England) rather than others (America). A history of a previous ulcer may be obtained in some cases.

Pathology.—Carcinoma may involve any part of the stomach, even the entire organ, but the lesser curvature and the pylorus are the favorite seats. Welch's analysis of 1300 cases showed the pylorus to be the seat in 791 (60.8 per cent.), the lesser curvature in 148 (11.4 per cent.), the cardia in 104 (8 per cent.), the posterior wall in 68 (5.2 per cent.), the whole or greater part of the stomach in 61 (4.7 per cent.), the greater curvature in 34 (2.6 per cent.), the anterior wall in 30 (2.3 per cent.), the fundus in 19 (1.5 per cent.), and multiple tumors in 45 (3.5 per cent.). The carcinoma develops from the mucous membrane, and, as a rule, speedily involves the submucous and even the muscular and the serous coats of the stomach, and it may spread to adjacent tissues and give rise to metastases. The primary growth varies much in size in different cases—from a small tumor, 2 to 3 cm. in diameter, to involvement of the entire stomach. It may appear as an annular growth about the pylorus; as a circumscribed flat elevation, round, ovoid, or irregular in outline; as a more or less exuberant cauliflower or polypoid growth prone to necrosis; or as a diffuse infiltration of almost if not all of the stomach. Extension to adjacent tissues by continuity of structure may ensue; and metastases (usually by way of the lymphatics) to the liver, regional lymph nodes, omentum, and to more distant organs, occur frequently. Welch's analysis of 1574 cases showed metastasis to the lymph nodes in 551 cases, to the liver in 475, to the peritoneum, omentum, and intestine in 357, to the pancreas in 122, to the pleura and lung in 98, to the spleen in 26, to the brain and meninges in 9, and to other

parts of the body in 92. Necrosis and ulceration are common. Portions of the growth may be found in the vomit, and, being subjected to microscopic examination, facilitate the diagnosis in a doubtful case. Perforation occurs in 4 per cent. of the cases, but it is usually preceded and frequently prevented by dense adhesions that bind the stomach to the liver, gall-bladder, colon, abdominal wall, etc. In one of my cases a large portion (about 10 cm. in diameter) of what at first appeared to be the wall of the stomach was made up of the ulcerating and necrotic left lobe of the liver—the stomach wall at this place having entirely disappeared. Rarely fistulous communications between the stomach and the intestines, lungs, pleura, the exterior, etc., may become established. In carcinoma of the pylorus with annular constriction the stomach is usually dilated from obstruction, and in some cases it is much distorted and displaced; in diffuse carcinoma the stomach wall may be greatly thickened and the organ reduced in capacity; whereas in carcinoma of the cardia the esophagus is usually dilated and the stomach sometimes atrophied.

The common carcinoma of the stomach is the cylindrical-cell epithelioma, which may be of the scirrhus, medullary, or colloid variety. The so-called malignant adenoma (adenocarcinoma), or adenoma destruens, is less common; and the squamous epithelioma occurring at the cardia, and usually associated with carcinoma of the esophagus, is the rarest. Scirrhus carcinoma may involve the pylorus, causing an annular constriction and consequent dilatation of the stomach, or it may involve almost if not quite all of the stomach, causing an appearance much resembling sclerosis of the stomach and exhibiting little tendency toward ulceration. Medullary carcinoma occurs as a soft cauliflower-like mass that grows with rapidity and commonly ulcerates, causing hemorrhage, and may give rise to perforation. Colloid (or mucoid) carcinoma occurs as a more or less diffusely infiltrating gelatinous mass that grows with rapidity and involves all the coats of the stomach. Malignant adenoma for a time presents a fairly typical histological appearance, but exhibits malignant characteristics by its diffusely infiltrating tendencies. Secondary carcinoma of the stomach has been observed, but is rare.

Symptoms.—The symptoms of carcinoma of the stomach vary much in different cases. Several of the most extensive carcinomas of the stomach that I have seen gave rise to no suggestive symptoms during the life of the patient, and this latency is by no means uncommon. In other cases the symptoms suggest and are sometimes mistaken for pernicious anemia; or secondary manifestations, metastases in the liver, superficial lymph nodes, etc., attract attention, and the primary growth is overlooked. In the majority of cases, however, the diagnosis is readily made, although not always in the early stages. In many cases evidences of chronic gastritis precede and they usually accompany the symptoms characteristic of carcinoma. In Osler's series of 150 cases the symptoms at the onset were: Pain in 48, dyspepsia in 44, vomiting in 21, loss of weight in

13, difficulty in swallowing in 3, and tumor in 1; in 7 the symptoms suggested pernicious anemia, and in 37 the onset was acute. The most suggestive symptoms are chronic indigestion with persistent epigastric pain, and loss of weight and strength, occurring in a person past middle life. The epigastric pain is burning or gnawing in character, often severe, more or less constant, frequently increased by food (sometimes not for several hours), and may radiate to the back or shoulders. Anorexia, nausea, vomiting, and constipation are features of almost all cases, but they may be missed. In the early stages the vomiting is not marked, but later, especially if either orifice is involved, it becomes a conspicuous symptom and may recur several times a day. In carcinoma of the pylorus the vomiting may be delayed for several hours after the taking of food; in carcinoma of the cardia it usually occurs soon; whereas in carcinoma of the body of the stomach it may occur rarely if at all. The vomited matter varies somewhat in different cases. If there be associated dilatation of the stomach the vomit partakes of the characteristics of this condition. Usually it consists of undigested, or partially digested, and often fermenting foods, mucus, and sometimes blood. Blood occurs in one-half or more of the cases. In most cases the blood is small in amount, and partakes of the general characters of coffee grounds—the result of the action of the gastric juice on the hemoglobin. In some cases the hemorrhage is profuse and the blood as ejected is either bright red or dark red and clotted, depending upon the length of time it has remained in the stomach. Blood may also appear in the stools, with or without associated hematemesis.

The loss of weight and strength, while in the main progressive, is subject to interruptions—marked temporary improvement often attending the removal of the patient to a hospital, the advent of a new physician, regulation of the diet, attention to the appetite, and treatment of the co-existing gastritis. Well-marked anemia is a prominent feature of the disease and usually progresses concurrently with the loss of weight; but it is sometimes so extreme and disproportionate to the other features of the disease as to suggest pernicious anemia. Usually, however, the blood is of the chloranemic type—the erythrocytes 2,500,000 to 3,500,000 (being rarely less than 2,000,000, as is common in pernicious anemia); the hemoglobin varies, but in well-marked cases it is less than 50 per cent.; and the leukocytes are sometimes slightly increased, 12,000 to 18,000. The anemia, emaciation, prostration, and certain attendant phenomena, such as a peculiar sallowness and sometimes pigmentation of the skin, a distressed countenance, etc., comprise the well-known cachexia. In some cases there is slight irregular fever (101° F.), attributable to the anemia or to infection and suppuration about the carcinoma.

Examination of the abdomen reveals a visible and palpable mass in at least 80 per cent. of the cases some time during their course. This varies much in position in different cases, but is usually in the epigastrium, hard, nodular, and tender. In many cases, especially if it has formed

adhesions with the liver or gall-bladder, it descends on inspiration, and it is often extremely mobile, susceptible of displacement over a wide area. On account of the anatomical conditions, as a rule, only tumors of the pyloric end of the stomach are palpable. In some cases, especially of pyloric obstruction, visible peristalsis may be apparent, or if the tumor sufficiently large or the patient sufficiently emaciated, a pulsation be transmitted from the aorta may be visible.

Examination of an Ewald test breakfast reveals absence of free hydrochloric acid in a large proportion (90 per cent.) of cases, and diminution in the amount of combined hydrochloric acid; but the total acidity, attributable to aromatic (lactic and other) acids, is often high. The presence of lactic acid, especially following preliminary lavage and a Boas' test meal, possesses much diagnostic value. The presence of a considerable or normal amount of free hydrochloric acid in the presence of symptoms suggesting carcinoma is sometimes believed to indicate ulcus carcinomatosum. Microscopicly the most important ingredients of the gastric contents are undigested food, yeast fungi, sarcinæ, blood (corpuscular or crystalline forms), and the Oppler-Boas bacillus—a long non-motile organism, usually of much diagnostic value, and answerable for the formation of the lactic acid. The milk-curdling ferment is usually reduced in amount, and the motor power of the stomach is deficient. The stools may contain large numbers of Gram-positive (Oppler-Boas) bacilli, in contrast to other conditions in which the stools are Gram-negative.

As the disease advances cachexia increases and metastatic nodules develop in different organs. Some of these are accessible to clinical observation and sometimes give a clue to the diagnosis—thus nodules in the liver, about the umbilicus, or in the left supraclavicular lymph nodes just external to the attachment of the sternomastoid muscle, etc. Perforation of the wall of the stomach with consequent peritonitis, or fistulous communications with different organs, may occur. Ascites due to carcinomatous involvement of the peritoneum or to compression of the portal vein by enlarged lymph nodes, jaundice due to obstruction of the bile ducts, and œdema of the legs due to anemia or thrombosis of the veins, may develop.

Diagnosis.—In the early stages the diagnosis is often in doubt, but chronic indigestion, persistent epigastric pain, loss of flesh and strength, absence of free hydrochloric acid and the presence of lactic acid in a test meal, occurring in a person past middle life, are extremely suggestive of carcinoma. After the occurrence of hematemesis and a palpable tumor the diagnosis is no longer in doubt; but at this time the disease has advanced to such a stage that hope of lasting benefit can no longer be entertained: the growth has made much local progress, and metastases undoubtedly have occurred. Chronic gastritis may be distinguished by the absence of cachexia, of profound anemia, of leukocytosis, and of lactic acid, the presence of free hydrochloric acid (although it may be temporarily absent), and the persisting improvement following treat-

ment. Gastric ulcer may usually be distinguished by its occurrence in younger subjects, by gastralgic paroxysms (pylorospasm), epigastric pain often augmented immediately by taking food and relieved by vomiting, persistent hyperchlorhydria, often profuse hematemesis (occurring in apparent health, as contrasted with the hematemesis of carcinoma which occurs in weakened and emaciated subjects), absence of cachexia, and absence of tumor (although it is sometimes almost if not quite impossible to differentiate between the induration and cicatrization of an ulcer and a carcinoma). Pernicious anemia may usually be excluded by the predominance of the gastric symptoms; by the fact that the erythrocyte count is rarely less than 2,000,000, and there may be a slight leukocytosis; and the characteristic blood picture of pernicious anemia (megalocytes, megaloblasts, etc.) is absent. Hypertrophic stenosis of the pylorus sometimes gives rise to confusion because of a mass in the epigastrium, but it occurs in younger subjects, there is no cachexia and no hematemesis, and the free hydrochloric acid is normal or increased. A gastrocolic fistula is suggested by fecal vomiting and the passage through the rectum of unaltered food; but fecal vomiting may be due to reversed peristalsis, and an extremely foul vomiting, scarcely to be distinguished from fecal vomiting, may be due to ulceration or gangrene of the carcinoma.

Prognosis.—Carcinoma of the stomach leads inevitably to a fatal termination—usually within a year from the time the disease is recognized. Some patients die within three months, and occasionally a patient lives two years from the time the disease is recognized.

Treatment.—The physician's chief concern, since he is unable to cure carcinoma of the stomach, should be to recognize it at the earliest possible moment, in the hope that prompt resort to surgical intervention may prove of lasting benefit. And since it is impossible in most cases to diagnose the disorder until such time that all hope of permanent relief has passed, he should suspect it in all cases of persisting indigestion, with lessened or absent hydrochloric acid and loss of flesh and strength, occurring especially about the fortieth year. In these cases, unless some other organic disorder can be found to explain the symptoms, the physician should advise an exploratory operation. This will enable the surgeon to make a positive diagnosis, and should the condition prove to be carcinoma, operation may be undertaken at a time when hope of success can be reasonably entertained. Aside from operative intervention, we are limited to relieving the patient's suffering, to feeding him, and prolonging his life. In many cases milk is the best diet; in other cases a mixed diet—milk, animal, and vegetable. Lavage is serviceable in persistent vomiting and in pyloric obstruction. Pain must be relieved by external applications, and by the internal use of alkalies, lime water, codeine, cocaine, morphine, or cannabis indica, and by the use of antifermentatives, such as carbolic acid, creosote, resorcin, salicylic acid, bismuth, and hydrochloric acid. The appetite may be stimulated by nux vomica and the bitter tonics (gentian, quassia, condurango, etc.).

NON-CARCINOMATOUS TUMORS.

Non-carcinomatous tumors of the stomach are rare and of pathological rather than clinical interest. Lipomas, fibromas, leiomyomas, sarcomas, cysts, and polyps (polyadenomas) have been described. Foreign bodies, of which the most curious and remarkable are hair balls, or hair tumors, occurring in hysterical women, have been mistaken for carcinoma.

HYPERTROPHIC STENOSIS OF THE PYLORUS.

Hypertrophic stenosis of the pylorus may occur as a congenital or an acquired condition. The congenital cases are sometimes due to ulceration or fissures at the pylorus; in other cases there seems to be only a spasmodic contraction of the pylorus (Thomson). The acquired cases consist of hyperplasia of the submucous and muscular coats of the pylorus. Some of these cases are perhaps developed upon a congenital basis, since they become manifest in adolescence. The *symptoms* consist of pylorospasm, pyloric obstruction, consequent dilatation of the stomach, and vomiting. In some cases there is visible peristalsis and a palpable mass. Some of the cases of periodic or cyclic vomiting in infants and children are believed to be due to this hypertrophic stenosis of the pylorus, and the associated diaceturia to be the result of starvation. In the mild cases, particularly those occurring in infants and children, benefit and often cure follows regulation of the diet, especially small amounts at frequent intervals of a bland and alkaline diet, and the use of alkalies and sedatives, such as the bromides, etc. In some cases resort must be had to operative intervention—dilatation of the pylorus, Loretta's operation, gastro-enterostomy, etc.

DISEASES OF THE INTESTINE.

Pathological Physiology.—The small intestine is designed for the further digestion of the chyme received from the stomach and for the absorption of the final products of this digestion. In the first portion of the duodenum the chyme is still acid in reaction (free hydrochloric acid)—which is undoubtedly one reason why the peptic ulcer of the duodenum is almost always found in close proximity to the pylorus. Lower down the acid chyme encounters and is rendered alkaline and otherwise acted upon by the bile and the pancreatic juice. Bile, of course, is secreted more or less continuously, but when the stomach is empty (that is, during the periods between digestion) it backs up in the gall-bladder, largely on account of closure of the sphincter at the end of the common bile duct. The stimulus to the discharge of bile into the duodenum seems to be proteins and fats, or some of their digestive

products; acids, alkalies, and starches are ineffective (Bruns). There is some reason, however, to believe that the secretion of bile may be stimulated, as is the pancreatic juice, by secretin. The proteins and fats entering the duodenum, excite, probably by a reflex action, a contraction of the gall-bladder, and an inhibition of the sphincter closing the opening into the intestine (Howell). Bile is both an excretion and a secretion. As an excretion it serves to eliminate some end-products of katabolism, such as cholesterin, lecithin, bile acids, and bile pigments; the bile acids and the bile pigments, however, may be in part reabsorbed. The chief action of the bile as a secretion is exerted in the breaking up and the absorption of fats. On account of its contained bile acids it serves as a medium for the suspension and splitting up of fats into fatty acids and glycerin, a hydrolysis effected by the lipase of the pancreatic juice materially aided by the bile. When bile is absent from the intestine the fat in the feces is greatly increased (from the normal 7 to 10 per cent. to as much as 80 per cent.); this is, in part, the cause of the pale color of the feces in jaundice, and it suggests the wisdom of withholding fats from the feces in such cases. The bile also serves to neutralize the acidity of the gastric chyme ard thus enable the trypsin of the pancreatic juice to act, trypsin being active only in an alkaline medium. Formerly the bile was supposed to exert an antiseptic action in the intestine, preventing putrefaction; but these is little real proof of this (the bile acts as a good culture medium for bacteria); its absence may permit of the putrefaction of proteins and fats which, were the bile present, would not occur. The bile was also thought to stimulate peristalsis, and to its absence the constipation of jaundice was attributed; but there is some doubt regarding the stimulating effect of the bile on peristalsis, and constipation is not always present in jaundice.

The secretion of the pancreatic juice is called forth by a chemical stimulus exerted by the acid chyme on the duodenal mucosa; this results in the production of a substance, secretin, the consequence of activation of an antecedent substance (prosecretin) by the acid of the gastric juice. Secretin is absorbed by the blood and carried to the pancreas, where, acting as a hormone, it stimulates the pancreas to activity. There is some reason to believe that other substances, fats, for instance, may act as the efficient stimulus, since the pancreatic juice seems to be secreted even in cases in which hydrochloric acid may be absent from the gastric juice.

The pancreatic juice contains three enzymes: (1) Trypsin, a proteolytic enzyme, which, however, is secreted by the pancreas as trypsinogen and is activated, converted into trypsin, by enterokinase, a product of the intestinal mucosa and a constituent of the intestinal juice. (2) Amylopsin (amylase), a diastatic enzyme, the action of which is similar to, if not identical with, that of ptyalin of the saliva; it serves to complete the digestion of starches. (3) Steapsin (lipase), a fat-splitting enzyme. Absence of pancreatic juice from the intestine results in deficient proteolysis and fat-splitting. The deficient fat-splitting leads to the occur-

rence of large, pale, grayish, slate-colored, or clay-colored, pasty, fetid, acid stools—which are quite characteristic.

The intestinal juice (succus entericus) also is active in digestive processes; but more so certain enzymes resident in the cells of the intestinal mucosa. These enzymes perhaps may be secreted into the intestinal juice, but some of them doubtless exert their activity within the epithelial cells themselves. These enzymes are: secretin, the hormone stimulating the pancreas to activity; enterokinase, which activates the trypsinogen of the pancreas, and forms the proteolytic ferment trypsin; erepsin, also a proteolytic enzyme, believed to assist in tryptic digestion; nuclease, an enzyme believed to aid in the digestion of nucleins; and certain inverting enzymes (maltase, invertase, and lactase) that connect disaccharids (maltose, saccharose, and lactose) into monosaccharids (dextrose, levulose, and galactose).

The stimulus calling forth the secretion of this succus entericus is not definitely known; it has been attributed to secretin and to the mechanical influence of the food. Mechanical and other irritants, such as mercury, arsenic, Bacillus dysenteriæ, saline cathartics, etc., induce a watery secretion, that differs materially from the normal intestinal juice; a similar watery secretion occurs in chronic passive congestion of the intestine, amyloid disease, etc.

The intestine always contain more or less gases, especially nitrogen, derived, in part, from the swallowed atmosphere, in part from bacterial decomposition of proteins; carbon dioxide, from carbohydrate fermentation and the interaction of the gastric acids on the alkaline hepatic, pancreatic, and intestinal secretions; and hydrogen, sulphuretted hydrogen, and marsh gas, largely from the putrefaction of proteins.

The intestinal contents are passed along by peristaltic movements, which consist of contractions moving on from segment to segment, while the part first contracted gradually relaxes. In addition, there are movements of rhythmical segmentation, which have been well studied by Cannon, and seem to subserve the purpose of thorough and intimate mixing of the intestinal contents. The intestinal movements are not continuously progressive from the beginning to the end of the intestine, but segmental and pendulum-like, though ultimately they serve to pass the intestinal contents onward. Readily digestible food appears to traverse the small intestine in about four hours, and to reach the sigmoid colon in two or three hours more (Hertz). Exaggeration of the intestinal movements gives rise to borborygmi, which is especially marked when the intestine contains much gas and little food. Exaggerated contractions against a mechanical obstruction or incoördinated contraction and relaxation give rise to intestinal colic; stimulation of the splanchnics (through which a nervous control is exerted) may cause colic and constipation (as in lead poisoning); and paresis or paralysis may lead to a form of nervous diarrhœa. Antiperistalsis does not occur in health, but may be observed in diseased conditions, especially those attended by intestinal obstruction.

The function of the large intestine is largely to absorb water from the intestinal contents, and thus indirectly to cause inspissation of the intestinal contents. Very little nutritive matter is absorbed by the colon, so that persons fed by rectal alimentation soon begin to suffer from starvation.

In health, divers sorts of bacteria are found in the intestinal tract, especially in the large bowel, notably Bacillus coli; in disease the number of species may become increased, or the normal inhabitants may be overcome by the invaders. Zoöparasites also are common. It seems likely that bacterial activity is of much importance in the orderly performance of the intestinal functions, and that derangements of this bacterial activity may lead to diseased processes. Those to which chief importance is attached are associated with putrefactive changes; the disorders to which they are supposed to give rise are called intestinal auto-intoxications, which have already been discussed (page 303).

The feces consist of: (1) Indigestible material (ligaments of meat, cellulose from vegetables); (2) undigested material, such as fragments of meat, starch, or fats which have escaped digestion; (3) products of intestinal secretions; (4) products of bacterial growth, especially indol and skatol, to which the odor of the feces is largely due; (5) cholesterin, probably derived from the bile; (6) purin bases, especially guanin and adenin; (7) mucus and epithelial cells thrown off from the intestinal wall; (8) pigment, especially urobilin or stercobilin, derived from the bilirubin of the bile; (9) inorganic salts—salts of sodium, potassium, calcium, magnesium, and iron, chiefly the last three together with phosphoric acid; and (10) microörganisms, usually in great quantities (Howell). Study of the feces is of much importance in determining the functional efficiency of the intestines. This is best done after the use for three or more days of Schmidt's test diet (see intestinal indigestion).

SYMPTOMATIC DISORDERS OF THE INTESTINE.

Disorders of the functions of the intestine are the natural consequence of definite anatomical lesions of most diverse nature, and as such are usually recognized and correctly ascribed to a more or less obvious cause. In other cases intestinal symptoms are due to, or part and parcel of, a general neurosis, such as hysteria, neurasthenia, etc., or they are reflex manifestations of disease of some adjacent viscera; whereas in other cases the intestinal disorder seems to be due entirely to some perversion of the functional activity of the intestinal nervous mechanism, and is unassociated with discoverable anatomical lesions; in other words, the condition is a neurosis. The dividing line between these different classes of cases, however, is by no means distinct, and sometimes cannot be drawn at all. We may, nevertheless, as in the stomach, distinguish disorders of motility, of sensibility, and of secretion, though they often do not occur alone.

Motor Disorders.—Disorders of motility are dependent upon increased or decreased irritability of the motor nerves and musculature: on the one hand, peristaltic unrest, nervous diarrhœa, or enterospasm, results; on the other hand, paresis or paralysis.

Peristaltic unrest (intestinal tormina), a condition allied to peristaltic unrest of the stomach, is not infrequently a manifestation of catarrhal enteritis or intestinal stenosis, but it sometimes occurs also as an apparently independent affection, especially in nervous, hysterical, and hypochondriacal women, and rarely even in persons without other neuropathic manifestations. The condition consists of rolling and gurgling sounds in the intestine, varying much in intensity from time to time, sometimes so loud as to be audible some considerable distance from the patient, and often occasioning extreme annoyance and discomfort, especially when they are, as sometimes, loud and squeaking. Pain is rarely complained of. The condition is due to increased peristalsis of the small intestine carrying along the gaseous and fluid contents from one segment of the intestine to another. Eructations may occur in associated peristaltic unrest of the stomach, and Rosenheim even reports feculent vomiting (which, however, must be exceedingly rare). The peristaltic movements are not infrequently visible and even palpable through thin abdominal walls. The attacks usually come on without notewor hy cause, though they may be provoked by emotional disturbances, certain articles of diet, etc.; they usually last for a few minutes, though sometimes for hours, and even during sleep; and they occur at irregular intervals.

Nervous diarrhœa is a condition dependent upon increased peristaltic movements, on the one hand, and an increased transudation of fluid into the intestine, on the other hand. Allied in some respects to peristaltic unrest, it differs in that, in at least some cases, the increased peristalsis seems to be almost, if not wholly, limited to the large intestine (whence the intestinal evacuations), and in the rapid transudation of fluid. This is certainly attributable to nervous influences (paresis or paralysis of the splanchnics)—since there is no other evidence of disease of the intestine, and the attack is almost always provoked by emotional disturbances, such as shock (unwelcome news), fright, ordeal of the witness stand, public speaking, examinations (students, etc.). A similar condition is sometimes observed in nervous disorders like tabes, and may occur as a reflex manifestation in different gastro-intestinal disorders, disorders of the female genitalia, e c. The stools vary in number from three or four to a dozen or more, are often passed in rapid succession, and consist of thin liquid material. Sometimes the first evacuation is of more or less well-formed fecal matter, whereas the subsequent stools are very thin and serous. Peristaltic unrest (rolling and gurgling sounds) sometimes accompanies the evacuations. A somewhat similar condition is occasionally observed in persons generally constipated, who when they become "nervous" from any cause, have an almost immediate evacuation of the bowels. Diarrhœa is also a common symptom of many diseases

of the intestine, notably acute and chronic enteritis, under which headings it is discussed.

Spasm of the intestine (enterospasm) is a term applied to tonic contracture of the muscular fibers (longitudinal and circular) of the intestine, resulting in lessening or complete though temporary occlusion of the lumen. The very rare occurrence of a primary or idiopathic enterospasm may be conceded, the subjects being neuropathic or hysterical individuals; the condition, however, is almost always secondary or symptomatic. It may be a manifestation of local inflammatory or other diseases of the intestine; it is a common manifestation of lead intoxication; and is not uncommon in diseases of the brain, especially meningitis, in which the intestinal contractures may be associated with scaphoid retraction of the abdomen. The spasm may be more or less diffuse, involving large sections of the small or large intestine, or both; or it may be quite circumscribed—of which variety, proctospasm (usually associated with other diseases of the rectum or anus) is a well-known example. Clinically the condition resembles enteralgia (of which it is really often the anatomical basis), and constipation is common; but neither pain nor constipation is essential. Local tympany or meteorism is not infrequent. When confined to the lower bowel, the feces may be narrow or ribbon-like, or resemble sheep's dung.

Paralysis of the intestine may be due to a variety of causes. It is common in peritonitis from whatever cause arisen; it may be a concomitant of intestinal obstruction; it is associated with a form of chronic atony with consequent constipation; and it is sometimes observed in hysterical subjects who suffer much from meteorism. Constipation is the conspicuous clinical feature. Paralysis of the anal sphincter is found in divers disorders of the brain associated with stupor and coma, especially infectious diseases like typhoid fever, various intoxications, and more purely local diseases of the brain associated with coma; it is a characteristic symptom of certain organic diseases of the spinal cord; and it sometimes develops after the persistence of irritating diseases of the rectum associated with long-standing tenesmus which ultimately leads to fatigue, paresis, and paralysis of the muscles. Paresis usually precedes paralysis, so that at first the patient by an effort can control the evacuations; later, the slightest disturbance or undue pressure results in the discharge of gas or fecal matter; and ultimately, when paralysis is complete, the intestinal contents are altogether beyond the control of the patient and may be discharged immediately they enter the rectum.

Treatment.—The treatment of the motor disorders of the intestine is similar to that of similar disorders of the stomach. Especial attention should be directed to the underlying conditions, whether neuropathic or local anatomical factors that may determine the onset, or augment an attack. The diet must be regulated and the bowels must be opened regularly and daily. Hydrotherapeutic measures, electricity, a course of treatment at certain health resorts, etc., are of benefit in many cases. Drugs, such as arsenic and the bromides, and sometimes strych-

nine and iron (in anemic conditions), may subserve a useful purpose. Belladonna or hyoscyamus may be useful in spasm; and physostigma or eserine may be useful in overcoming constipation due to atony.

Sensory Disorders.—The sensibility of the intestinal tract may be increased (hyperesthesia, pain), diminished (anesthesia), or perverted (paresthesia).

Intestinal pain (*enterodynia*) may be due to one of several causes: (1) It may be a manifestation of inflammation of any portion of the intestine and of its peritoneal covering (to be discussed under appropriate headings). (2) It may be the so-called intestinal colic. (3) It may be a true intestinal neuralgia.

Intestinal colic, the pain produced by violent tetanic contractions of the muscular coat of the intestine, may be due to: (1) Sudden chilling of the surface of the body, which of itself seems to provoke intestinal colic in certain susceptible persons; (2) dietetic indiscretions—coarse, indigestible, unripe, or decomposed food, especially unripe fruit and overripe fish, and cold drinks; (3) foreign bodies, such as gallstones, enteroliths, scybalous masses, large amounts of mucus (mucous colic), etc.; (4) drastic cathartics and other medicines; (5) infections and intoxications, such as certain infectious diseases (cholera), lead poisoning, etc.; (6) zoöparasitic invasions, intestinal worms; (7) reflex disturbances associated with disease of the ovaries, uterus, bladder, kidneys, etc.

Intestinal colic usually comes on suddenly with severe pain radiating in different directions (though it may remain localized); it varies much in intensity (from moderately severe "griping" to extremely severe pains), remits and intermits, and varies in duration from a few moments to several hours. Nausea, vomiting, flatulence, and faintness are not uncommon; if the pain is severe, cold perspiration usually breaks out; and exhaustion and collapse may supervene. The attack usually subsides with the removal of the exciting cause, often with the discharge of feces (stercoral colic), sometimes with the evacuation of gas (flatulent colic), or of mucus (mucous colic).

Intestinal colic may simulate hepatic and renal colic in the sudden onset and sudden cessation, as well as severity, of the pain, but in hepatic colic the pain is usually referred to the right hypochondrium, and may be referred to the right shoulder; it is not so likely to be intermittent; jaundice and bile-stained urine may occur; there may be a history of similar attacks; and gallstones may be found in the feces. In renal colic the pain is usually in the back, loin, or groin, and radiates to the testicle, which may be retracted; strangury or frequent micturition is common, the urine may become bloody, and a calculus may be found in the urine.

The term *intestinal neuralgia*, or *nervous enteralgia*, may be applied to attacks of abdominal pain, seen in nervous, hysterical, and hypochondriacal women (rarely men), which come on suddenly, spread usually from the umbilicus over the abdomen, last a variable time are likely to recur, and seem to be unusual in severity, leading fre-

quently to collapse, and in which there is no etiological or other evidence of true intestinal colic, nor of inflammation of the abdominal viscera or peritoneum. Vomiting is absent, the stomach and intestines perform their functions normally; but cutaneous hyperesthesia and other evidence of hysteria, etc., are present. In the absence of better knowledge, the condition is described as a neurosis—a neuralgia of the mesenteric plexus and the splanchnic nerves; but in some cases, as for instance the crises and other perhaps analogous conditions, the pain is doubtless due to angiospasm.

Diminished sensibility (anesthesia) of the intestine attends and may be the cause of constipation. It is found especially in different diseases of the brain and spinal cord associated with paralysis, and in fecal accumulations. If the motor innervator of the intestine is unimpaired, spontaneous evacuations occur, but with defective motility constipation, often extremely obstinate, occurs.

Paresthesia of the intestine is rather unusual, though it may be complained of by the hysterical and the insane. The paresthetic phenomena are usually (though not always) referred to the rectum.

Treatment.—The treatment of the sensory disorders of the intestine is determined by the etiological factor. The pain may be relieved by hot applications to the abdomen—poultices, hot-water bag, mustard plaster, spice poultice, turpentine stupe, and by the internal administration of spirit of ginger, aromatic spirit of ammonia, compound spirit of ether, camphor, chloroform water, or a dry Martini cocktail (the one instance in which it subserves a very useful purpose). Rarely morphine or other preparations of opium must be resorted to. The bowel should be evacuated immediately by rectal enemas, and by castor oil by the mouth. Subsequently attention must be directed to the diet, to regularity in the movement of the bowels, and to the underlying general condition. Tonics, stimulants, and other measures mentioned under constipation (page 656) are sometimes necessary to overcome the constipation due to diminished sensibility; in some cases manual removal of the feces may have to be resorted to.

Secretory Disorders.—The intestinal juice under the influence of the nervous system is subject to increase and decrease—depending upon a variety of factors. Diminution is doubtless a factor in some cases of constipation; increase is an important factor in so-called nervous diarrhœa, in which the evacuated fluid is largely serous in character; but the only disease described as wholly or almost wholly a secretory disorder or neurosis is so-called mucous colic or colitis.

INTESTINAL INDIGESTION.

(*Intestinal Dyspepsia; Dystrypsia.*)

In health the entrance of the chyme into the duodenum, by activating the prosecretin of the duodenal mucosa and thus forming secretin, gives rise to the secretion of bile and of pancreatic juice which not only

neutralize the acidity of the chyme, but also produce that alkalinity required for the proper digestive activity of the ferments contained in these juices. Intestinal digestion then depends not only upon the proper amount of bile and of pancreatic juice of good quality, as well as of intestinal juice, but also upon the functional integrity of the stomach: since defective gastric digestion not only results in the introduction into the duodenum of chyme ill adapted to the intestinal functions, but deficient or increased acidity results in corresponding derangement of the flow of bile and of pancreatic juice into the intestine—though doubtless minor deviations from the normal can be compensated for. Intestinal indigestion—disorders of the processes of digestion occurring in the intestine—may be primary or secondary, although it is often difficult, if not impossible to separate the one from the other.

Etiology.—Primary intestinal indigestion results from: (1) Disease of the small intestine, of whatsoever nature, but especially chronic catarrh, interfering with the absorptive power of the mucous membrane, as well as with the formation of an active intestinal juice. (2) Change in the quality or quantity of the bile. A diminished amount of bile may be due to sluggish action of the liver or to obstruction to the entrance of bile into the intestine arising from any cause whatever. Changes in the quality of the bile are ill understood, though it seems that the liver has the function of eliminating by way of he bile and the intestine certain end products of metabolism, and it is not inconceivable that these in excess may interfere with the action of the bile or irritate the intestinal mucosa. (3) Changes in the quantity or quality of the pancreatic juice. Secondary intestinal indigestion may arise in consequence of any form of gastric indigestion—especially gastric hyperacidity, but also subacidity, deficient motor power, etc.

Symptoms.—The symptoms of intestinal indigestion usually accompany and sometimes cannot be dissociated from those of gastric indigestion. The patient usually complains of loss of appetite, acid eructations, flatulency, epigastric distress and distention (in consequence of acid fermentation of carbohydrates, putrefactive decomposition of albuminous foodstuffs), and of diarrhœa, the result of irritation and consequent increased peristalsis often occasioned by abnormal acidity of the intestinal contents. The diarrhœa is often and characteristically preceded by pain, which may be colicky in nature. The stools at first consist of the contents of the lower part of the bowel, but soon present the characteristics of the contents of the jejunum, being semifluid, gelatinous, and tenacious—due to a large amount of mucus, the normal contents of the jejunum, and not the result of catarrh (as shown by the absence of formed elements, epithelial cells, and round cells so common in catarrh). Furthermore, the stools, often yield a pronounced reaction for bile (though this, of course, varies with the amount of bile present); they are sometimes quite greenish in color, though often light yellowish and foamy, and yield an odor of butyric acid.

The nature of the disorder in protracted cases is best determined by

study of the stools after the use for three days or more of Schmidt's test diet. This consists of: In the morning, 500 c.c. of milk and 50 grams of Zwieback; in the forenoon, 500 c.c. of oatmeal gruel (made up of 40 grams of oatmeal, 10 grams of butter, 200 grams of milk, 300 grams of water, 1 egg, and some salt); at noon, 125 grams of chopped beef broiled rare, with 20 grams of butter, and 250 grams of potato puree (190 grams of potatoes, 100 grams of milk, 10 grams of butter, and a little salt); in the afternoon, milk and Zwieback, as in the morning; and in the evening, oatmeal gruel, as in the forenoon. This diet furnishes about 2250 calories. Special attention should be paid to the presence in the stool of connective tissue, muscle tissue, potato (or starch) granules, fat (fatty acids and soaps), mucus, and bacteria. Special importance attaches to the presence of muscle tissue, starch granules, and fatty acids. Resort may also be had to Sahli's dermoid test, etc.

Persistence of the attack soon leads to catarrh of the intestine, and the usual catarrhal stools (alkaline reaction, evidences of catarrhal desquamation, etc.).

Diagnosis.—The cases due to disorder of the stomach may be distinguished by symptoms especially referable to the stomach, the results of analyses of the gastric contents after a Boas or Ewald test meal, the presence of much connective tissue in the stools after Schmidt's test diet, and improvement following treatment directed to the stomach.

Treatment.—The treatment consists especially in regulating the diet, in accordance with the findings of a gastric analysis and a study of the stools. The medicinal measures depend upon the underlying conditions and the digestive capacities. Gastric hyperacidity or subacidity calls for appropriate treatment. Carminatives and so-called intestinal antiseptics, such as resorcin, bismuth salicylate, naphthalene, creosote, carbolic acid, aromatic spirit of ammonia, salicylic acid, etc., are usually indicated. Calomel in small doses, $\frac{1}{40}$ to $\frac{1}{20}$ grain (0.0015 to 0.003 gram) three or four times a day, is an excellent remedy in some cases, from time to time, although, if it stimulates peristalsis, the dose should be reduced.

INTESTINAL HEMORRHAGE.

(*Enterorrhagia.*)

Etiology.—Blood issuing from the body by way of the anus may be evidence of hemorrhage from any part of the gastrointestinal tract, and may be due to many causes—local and general. The general causes are such as determine hemorrhage in other parts of the body—purpura, scurvy, malignant (or hemorrhagic) forms of certain specific infections (smallpox, yellow fever, cholera, malaria, measles, scarlatina, diphtheria, etc.), septic infections (malignant endocarditis, etc.), leukemia, pernicious anemia, etc.; and it is rarely a manifestation of vicarious menstruation. The local causes are: (*a*) Hyperemia of the intestine, common in advanced cirrhosis of the liver, and in heart disease, less common in the

early stages of acute inflammations, such as typhoid fever. (*b*) Inflammation, erosion, and ulceration of the intestine, such as the inflammation provoked by irritant poisons or drastic cathartics; peptic or other ulceration of the stomach or duodenum; and the various forms of ulceration (simple, tuberculous, syphilitic, carcinomatous, etc.) of other portions of the intestine; typhoid fever; dysentery; etc. In some cases even large hemorrhages seem to have resulted from mere superficial erosion of the mucous membrane. (*c*) Embolism and thrombosis of the mesentric vessels. (*d*) Changes in the vessel walls, such as occur in amyloid disease, nephritis, etc., may occasion hemorrhage—in some cases in consequence of the formation and rupture of miliary aneurysms. (*e*) Zoöparasitic invasion of the intestine, as by Uncinaria duodenalis, Schistosoma hæmatobium, etc. (*f*) Hemorrhoids, internal and external, and anal fistula or fissure. (*g*) Trauma, external blows, or internal injuries provoked by divers kinds of foreign bodies (chicken bones, oyster shells, enteroliths, hard feces, etc.). In women, blood supposed to issue from the bowels sometimes comes from vaginal sources. In addition, blood issuing from the anus may be due to swallowing of blood coming from the nose, throat, œsophagus, or lungs (hemoptysis).

Symptoms.—The symptoms of intestinal hemorrhage vary much with the cause and the amount of the hemorrhage. When slight, the hemorrhage often passes unnoticed, or may be merely an accidental finding in the stool. This is sometimes the case in latent ulceration, carcinoma, or parasitic invasion of the intestines (occult blood), and may require microscopic or chemical tests for its detection. When large the symptoms may be exceedingly severe, and occasionally lead directly to death, although as much as two or three quarts may be lost at one time, and even a debilitated patient survive (typhoid fever). Sudden large hemorrhage is manifested by faintness, dimness of vision, and vertigo, often progressing to collapse, pallor of the skin and visible mucous membranes, profuse perspiration, and fall of temperature (often 5° to 8° or more). Repeated small hemorrhages lead to the gradual development of marked anemia (see chronic secondary anemia, page 367).

When the blood comes from the stomach or the upper part of the intestine it is usually altered by the digestive juices, and issues from the anus as black tarry fluid or masses (melena). Very rarely a massive hemorrhage coming from the stomach or upper part of the duodenum and issuing soon from the anus may be but little altered. Blood coming from the lower part of the small bowel, or from the colon or the rectum, is comparatively unaltered and readily recognized as blood. Should the hemorrhage occur from the stomach, or from the upper part of the intestine, and the blood find its way into the stomach, hematemesis may accompany the enterorrhagia. But very large, and even fatal hemorrhages may occur without any external discharge of the blood.

Treatment.—The treatment of intestinal hemorrhage is to be based entirely upon its source. In general it is similar to that of hemorrhage occurring in typhoid fever (page 59).

MUCOUS COLIC.

(*Mucous Colitis; Pseudomembranous Enteritis; Tubular Diarrhœa.*)

Mucous colic is a secretory neurosis of the colon characterized by the formation of an excessive amount of tough, tenacious mucus.

Etiology.—Mucous colic is not so rare a disease as was formerly supposed, and is most common in neurotic and hysterical women (80 per cent. of the cases). It is rare in children. In most cases it is an independent disorder, and occurs without noteworthy anatomical lesions in the intestinal mucosa. In some cases, however, it seems to become engrafted upon an intestinal catarrh; that is, to the catarrhal alterations are added, either permanently or periodically, the manifestations of the neurosis (hypersecretion of mucus). Constipation appears to be a contributing etiological factor in some cases.

Symptoms.—The disease is characterized by the passage from the bowel of large amounts of mucus. This occurs usually in paroxysms, frequently of unknown cause, but sometimes induced by psychic disturbances, dietetic indiscretions, and divers extraneous causes. The paroxysm is usually attended by severe abdominal pain, which is sometimes so severe as to suggest to the patient impending dissolution, and to engender the morphine habit. The pain is most commonly along the course of the descending colon, and is frequently accompanied by tenderness. The pain and tenderness, however, may be on the right side, and being sometimes associated with palpable tenderness of the appendix, suggest an etiological relationship, which seems to be proved in some cases by recovery after removal of the appendix. Rarely pain is absent. Tenesmus is a distressing feature of some cases. The attack may last for from several days to two weeks or longer, but mucus is not passed with each paroxysm of even severe pain. The mucus occurs as shreds, flakes, narrow cylinders (suggesting certain intestinal worms), or sometimes as tubes forming veritable casts of the bowel. Usually the stool consists exclusively of this mucus, and a very large amount may be passed at one time. As a rule, the mucus is opaque and grayish; occasionally it is brownish from admixture with feces, and rarely reddish, from admixture with blood. Occurring in masses or as casts the mucus often resembles fibrinous exudates, but microscopic examination (mucus cells, cylindrical epithelial) and chemical examination (mucin reaction) reveals it to be composed exclusively of dense, tough, altered mucus.

As a rule, the patients present the ordinary manifestations of hysteria, neurasthenia, hypochondriasis, etc. Constipation is the rule between attacks. The condition lasts for years, and in many cases, ultimately leads to marked anemia and emaciation.

Treatment.—The treatment is eminently unsatisfactory. During the paroxysm measures should be taken to relieve the pain, such as hot fomentations to the abdomen, and to get rid of the cause of the pain by a thorough evacuation of the bowels—by castor oil and rectal enemas.

The cure of the disorder comprises attention to the underlying hysteria or neurasthenia, rather than measures suggested by a supposed intestinal catarrh (which is rarely present). Attention to the common constipation is frequently followed by marked improvement in the mucus colic. A right-sided colotomy with maintenance of an artificial anus for some time, and consequent rest of the colon, has been successful in a few cases.

CONSTIPATION.

(*Costiveness.*)

Constipation is a condition in which the feces are retained in the intestinal canal an unusually long time; as a rule, the feces when passed are unduly hard. There may be a daily movement, in which event the evacuation is insufficient.

Etiology.—Constipation may be due to many causes—general and local. The general causes comprise family or individual tendency, "bad habits" (non response to the calls of nature), sedentary life, dietetic errors (too little food, or foods that leave little residue), habitual use of purgatives, certain nervous disorders (such as hysteria and neurasthenia, as well as organic disorders interfering with the evacuation of the bowel), hepatic disorders associated with jaundice, acute infectious fever, general debility, etc. The local causes are: (1) Diminished expulsive power, that is, defective intestinal peristalsis, atony of the intestine (itself also a result of chronic constipation), or weakness of the diaphragm or the abdominal muscles (obesity, distention from repeated pregnancies, etc.); and (2) increased resistance to the onward movement of the feces, which may be due to (*a*) hardness and dryness of the feces; (*b*) lessening of the intestinal lumen from any cause (see intestinal obstruction); (*c*) inhibition of the free movement of the intestine in consequence of adhesions or abdominal bands; (*d*) displacement of the intestine (V-shaped colon, redundant sigmoid); (*e*) spasm of the intestine, usually of the lower colon or sigmoid (with atony and dilatation of the proximal parts of the intestine)—so-called spasmodic constipation, observed especially in hysterical and neurasthenic women, particularly the subjects of ovarian or uterine disease.

Constipation is symptomatic when due to obvious causes, idiopathic when no cause can be ascertained. When symptomatic, it is usually more or less temporary; when idiopathic, it is not infrequently a habit—so-called habitual constipation.

Symptoms.—There is marked diversity in the reaction to constipation. Many persons are habitually constipated and otherwise apparently well; others, occasionally constipated for greater or less periods, suffer little discomfort; whereas in others the failure to have the daily evacuation produces immediate and notable discomfort, and protracted constipation brings a long train of symptoms in its wake—copremia (intestinal auto-intoxication). Most extraordinary cases are occasionally

observed—cases in which no evacuation has occurred for weeks and even months.

Although, in many cases, constipation is the only complaint, there is often a feeling of discomfort in the rectum. Overloading and consequent distention of the sigmoid may occasion pain from pressure on regional nerves. There is frequently associated flatulence, borborygmi, occasional attacks of colicky pain, and diarrhœa (which should not be misinterpreted). Hemorrhoids are common; inflammation, dilatation, ulceration, and even perforation of the intestine may result; and menstruation is likely to be painful. The retained feces usually become hardened, in consequence of absorption of the watery constituents, and form scybalous masses; these may be palpated through the abdomen as irregular, sometimes sausage-shaped tumors, in the course of the colon. Occasionally scybalous masses retained in sacculi of the colon give rise to the development of enteroliths. In spasmodic constipation the fecal discharges may be narrow and ribbon-like, or resemble the discharges of sheep.

In many cases the appetite is lost (or lessened), the tongue coated, and the breath foul; headache, vertigo, palpitation of the heart, are not uncommon; and depression of spirits, hypochondriasis, etc., are likely to supervene.

Constipation in infants may be congenital, or due to dietetic indiscretions, or to local causes similar to those causing constipation in adults. Some infants, constipated from birth, require for months a daily enema or aperient to secure an evacuation. Certain artificial foods seem to engender constipation in some infants, a deficiency of fat being believed to be the active factor. In older children the costive habit is often favored by inattention on the part of the mother.

Treatment.—The cultivation of habits of regularity is of the utmost importance, and only those who have observed know the good effects that may be thus achieved. The patient should go to stool at the same time every day, whether there is a desire to evacuate the bowels or not, and every such desire should be immediately gratified. The diet should comprise considerable fruit and vegetables (which leave a residue). A glassful of cold water before breakfast, an orange or oatmeal at breakfast, and stewed fruits and salads at dinner subserve a useful purpose in many cases. Persons of sedentary habits are often benefited by exercise; abdominal massage is useful in some cases; and an abdominal binder is of value to those with a pendulous flabby abdomen and visceroptosis.

Drugs should be dispensed with as long as possible. Even in long-standing cases much and lasting benefit may be secured by dietetic and hygienic measures. Medicinal measures when necessary vary with the nature of the causal factor, which must be diligently searched for. At the beginning of the treatment it is often advisable to clear the intestine thoroughly with castor oil, a blue mass pill, or calomel, followed by a saline aperient. In many cases, the best results are obtained by a

daily injection of tepid water with or without soap; in other cases injections of oil are much better; but enemas should not be too long continued. Some patients are much benefited by a saline aperient water, sodium phosphate, or other saline, taken a half hour before breakfast. Those who are occasionally constipated, find relief in a Seidlitz powder, compound licorice powder, the official compound cathartic pill, or a glycerin suppository. Should a course of medicine be necessary, the desired results may usually be secured by the use of cascara sagrada, which has the advantage that, having been continued for some time, the dose necessary to secure a daily evacuation may be gradually reduced, and the drug ultimately dispensed with, should the patient continue habits of regularity. The pill of aloin ($\frac{1}{8}$ grain, 0.008 gram), strychnine ($\frac{1}{40}$ grain, 0.0015 gram), and extract of belladonna ($\frac{1}{10}$ grain, 0.006 gram), though much abused, is very useful in many cases. The following pill night and morning is also useful:

℞—Podophyllin	$\frac{1}{10}$ to $\frac{1}{5}$ grain	0.006 to 0.012
Strychnine	$\frac{1}{40}$ grain	0.0015
Ext. physostigma	$\frac{1}{8}$ grain	0.008
Ext. belladonna	$\frac{1}{6}$ grain	0.01

Instead of the podophyllin, aloin, extract of cascara sagrada, or compound extract of colocynth may be substituted. Phenolphthalein (1 to 2 grains, 0.06 to 0.12 gram) at night is often attended by good results. Rhubarb and sodium bicarbonate also are of value in some cases. A pill of iron (dried sulphate of iron, 2 grains, 0.12 gram) and extract of aloes (1 grain, 0.06 gram), with or without rhubarb (5 grains, 0.3 gram), is often efficacious in the constipation of anemic and chlorotic conditions. Agar-agar (15 to 30 grains, 1 to 2 grams or more) eaten with cream, twice or thrice daily, is often serviceable when the feces are hard and small in amount. Similar results may be achieved by the use of large amounts of fats, olive oil, butter, cream, etc.

In children the best results follow attention to the diet, the giving of cream, barley water, oatmeal water, etc. Rectal injections may be required, or a conical piece of soap may be introduced within the anus. Among medicines, magnesia, manna, rhubarb and soda, and olive oil are probably the best, but if possible all medicine should be avoided.

ACUTE ENTERITIS.

(Acute Intestinal Catarrh; Cholera Morbus; Cholera Nostras.)

Acute enteritis is an acute inflammation of the intestinal mucous membrane, characterized by abdominal pain and diarrhœa.

Etiology.—The affection occurs at all ages, in both sexes, and is especially prevalent during the summer months. Its causes are: (1) Bacterial infection, which sometimes causes a primary infectious enteritis. An enteritis is common also in many infectious diseases, such as typhoid fever, cholera, pneumonia, dysentery, etc. (2) Animal para-

sitic infections—worms, etc. (3) Toxic and mechanical irritants, such as improper food (tainted meats, overripe or underripe fruit); stomach contents improperly prepared for the intestine; intestinal contents that remain too long in the intestine (in consequence of stenosis or imperfect action of the intestinal musculature); drastic cathartics; irritant poisons; certain articles of diet and waters to which the individual may be unaccustomed (personal idiosyncrasy), etc. (4) Certain autogenic poisons, such as occur in uremia, and which irritate the intestinal mucosa. Secondary enteritis occurs in heart disease and liver disease in consequence of passive congestion; in amyloid disease (undue permeability of the intestinal bloodvessels), etc.

Pathology.—The lesions are those of catarrhal inflammation in general—swelling, hyperemia, and increase of mucous secretion, with desquamation of the surface epithelium. In many cases, however, at the necropsy, but few or slight changes may be apparent. Frequently, in addition to swelling, there is superficial erosion or ulceration of the solitary and agminate lymph follicles (catarrhal and follicular ulceration). In severe cases, small hemorrhages in the mucosa may occur (hemorrhagic enteritis). The lesions may extend throughout the entire intestinal tract, but are often more marked in one part than another.

Symptoms.—Diarrhœa may be the only symptom, and even this may be inconspicuous in catarrh of the upper part of the small bowel. The stools vary much in frequency—four to twenty or more in the twenty-four hours; in amount, from small movements to great losses of fluid (liters); in color, from dark brownish to light yellowish or grayish, depending upon the amount of biliary coloring matter; and in consistency, depending upon the amount of watery constituents and the increased peristalsis hurrying on the fluid intestinal contents. Diarrhœa characterized by the evacuation of much fluid is spoken of as serous; that in which there is much fecal matter, as stercoral; that in which the stools contain undigested food, as lienteric; and the profuse serous discharges occurring in and adding to the exhaustion of the last stages of severe diseases, such as tuberculosis, etc., as colliquative diarrhœa. Mucus is always present in greater or less amount, and microscopicly one may detect mucus, desquamated epithelial cells, particles of food (muscle fibers, starch granules, etc.), crystals of calcium phosphate and oxalate, cholesterin, and different microörganisms.

In most cases there is more or less abdominal distress, which often amounts to severe, sometimes colicky, pain, and is often marked just before a stool. There is often also some abdominal distention, tympanites, borborygmi, and tenderness, and in case the lower colon be involved, tenesmus. The appetite is lessened or lost, the tongue is coated, thirst may be marked, and rarely there is vomiting (severe cases). The urine is lessened, sometimes greatly lessened, and may contain a little albumin, and indican and ethereal sulphates in excess. There may be, though usually there is not, slight fever.

In very severe cases (primary infective enteritis) the pain may be

marked, vomiting severe, and the stools many and copious (resembling cholera: cholera nostras, cholera morbus); cramp-like pains in the calf muscles is common; and collapse attended by heart failure, cold extremities, somnolence, and delirium may supervene. Many of these are undoubtedly cases of ptomaine or other food poisoning.

Diagnosis.—The diagnosis is usually easy, though several days' observation is sometimes necessary to exclude certain infectious diseases—typhoid fever, dysentery, etc. Especial involvement of the small intestine is suggested by less frequent stools, borborygmi, colicky pain about the umbilicus, greenish or yellowish stools rather intimately mixed with mucus and containing particles of undigested food (lienteric stools), and the fact that a stool is often induced by taking food. Involvement of the large bowel is suggested by pain along the course of the colon, tenesmus, frequent fluid, "soupy," grayish stools, containing large flakes of mucus. Duodenitis is suggested by the development of jaundice (from the extension of the catarrhal inflammation to the bile duct).

Prognosis.—The prognosis in general is good, the ordinary cases resulting in a cure in from three to five days. The manifestations, however, sometimes become chronic. The prognosis is often very serious in the infective form, as well as in debilitated subjects (tuberculous, cardiac, hepatic, and nephritic lesions). Children and debilitated subjects often die.

Treatment.—The prime indication in all cases is to rid the intestine of all irritating substances—whence an initial purgative is called for in almost all cases, even in many cases in which the evacuations have been numerous. Mild cases respond well to warm applications to the abdomen, 1 ounce (30 c.c.) of castor oil with 20 minims (1.25 c.c.) of tincture of opium, or calomel in repeated small doses, and restriction of the diet (milk, whey, and toast) for several days. It is well to give in addition intestinal antiseptics and astringents, such as the bismuth, salol, and chalk mixture (page 148), or a lead and opium pill (page 149). Resorcin, creosote, beta-naphthol, bismuth, and similar preparations are also useful. Involvement of the large bowel is best treated by cleansing enemas (to which boric or salicylic acid may be added), followed by a starch enema with tincture of opium. Depression is to be treated by stimulants, hot tea with whiskey or cognac, camphor, etc. The extreme depression that sometimes attends many serous evacuations, as well as the evacuations themselves, are often promptly controlled by morphine, $\frac{1}{4}$ grain (0.015 gram). Tonics are indicated during convalescence, and the diet must be regulated.

CHRONIC ENTERITIS.

Etiology.—Chronic enteritis is due to: (1) The repeated or long-continued action of the factors causing acute enteritis; (2) protozoan infection (amœba trichomonas, cercomonas, balantidium, etc.); and (3)

it may occur secondarily to diseases of the heart, lungs, stomach, liver, and kidneys.

Pathology.—The lesions are similar to those found in chronic gastritis, and are most frequent and marked in the lower part of the ileum and the colon. They consist of: (1) Hypertrophic processes—thickening of the mucosa (with the formation of much mucus), sometimes going on to polypoid masses, swelling of the solitary lymph nodes and plaques, follicular ulceration; and (2) atrophy—of the glandular tissue, with cicatrization, and consequent thickening of the intestinal wall. These two processes are often observed side by side. The follicular ulceration sometimes leads to phlegmonous infiltration of the intestinal wall (in consequence of secondary infection); and in consequence of healing and cicatrization, stenosis sometimes results.

Symptoms.—Several groups of cases may be distinguished: (*a*) In a minority of the cases the symptoms are much like those of the acute catarrh, although more mild. The patient has several diarrhœic stools daily, attended by colicky pains and borborygmi, and in the event of involvement of the large intestine (as is usual) with tenesmus. The diarrhœa is extremely obstinate, especially in the event of ulceration, though even without it. In consequence of the common ulceration, blood and pus, and even particles of the necrotic tissue, may be found in the stools. (*b*) More common than diarrhœa is constipation, the consequence of atony of the intestinal musculature and comparative insensibility of the nerves of the mucosa. (*c*) In perhaps the majority of cases, periods of diarrhœa alternate with periods of constipation. During the period of constipation a thick mucus is secreted, feces accumulate, fermentative processes go on, the intestine becomes distended, and these culminate in such irritative processes as to result in rather marked serous evacuations. In some cases considerable mucus is secreted, and, becoming coagulated, forms masses or veritable casts of the intestines. In most cases well-marked nervous symptoms develop—apathy, headache, palpitation of the heart, somnolence, hypochondriasis, etc. Cutaneous manifestations (urticaria, pruritus), as well as tetany, attributable to auto-intoxication, are rarely observed. The patient frequently emaciates considerably, and a high grade of secondary anemia may develop.

Diagnosis.—It is desirable to exclude specific ulceration—tuberculous, syphilitic, dysenteric, carcinomatous; as well as certain neuroses.

Prognosis.—The prognosis is doubtful in many cases. In children and in the aged it is very bad.

Treatment.—The treatment in general is similar to that of acute enteritis, but the strictest attention must be paid to dietetic and hygienic measures. In cases marked by alternate diarrhœa and constipation, special effort must be made to cure the constipation, since this is always an irritating factor. A clue to the correct diet may often be obtained by a study of the stools after the use of the Schmidt test diet (page 652). In some cases no improvement follows until the patient is put upon the strictest dietetic regimen, whence the good results often attained

at different spas and health resorts. The chronic lesions of the diseased mucosa are very resistant to medicinal measures, but certain good results seem sometimes to follow the administration of silver nitrate, lead acetate, zinc sulphate, bismuth preparations, naphthalene, tannic acid and drugs containing tannic acid. In involvement of the large bowel irrigation is useful, good results attending the use of sodium chloride solution, 0.6 per cent.; silver nitrate, 0.02 to 0.05 per cent.; boric acid, 0.5 per cent.; salicylic acid, 0.1 per cent.; tannin, 0.2 to 0.5 per cent.

DIARRHŒAL DISORDERS OF CHILDREN.

(Summer Diarrhœa of Children; Gastro-enteritis of Children; Acute Gastro-intestinal Catarrh; Acute Dyspeptic Diarrhœa; Gastro-intestinal Intoxication; Cholera Infantum; Enterocolitis; Ileocolitis; Infantile Dysentery.)

Etiology.—Diarrhœal disorders are exceedingly common during the summer months (June, July, and August, but also May and September) among children, from the sixth to the eighteenth month. While especially common among children of the poor, occurring frequently in tenement houses and asylums, they attack also those reared under the most favorable auspices. The special etiological factors are associated with diet, atmospheric temperature (summer heat), bad hygiene, and general inattention. Predisposing factors comprise feeble constitution, malnutrition, marasmus, rickets, etc. According to Holt, only 3 per cent. of the fatal cases occur among breast-fed infants.

The mild cases, so-called, acute dyspeptic diarrhœa, result from over feeding, too frequent feeding, the use of improper food, and the sudden change of food (such as may occur in weaning), and they are predisposed to by the heat and humidity of summer, fatigue, acute infections, etc. The feces usually contain the normal intestinal bacterial flora, no abnormal types being found with sufficient frequency to be considered of etiological significance. More severe cases, designated gastro-enteritis, gastro-intestinal intoxication, or cholera infantum, also result from the aforementioned causes; some of them are examples of acute milk poisoning, the bacteria, perhaps, having been destroyed by heat, but their toxins or ptomaines persisting; others are due to Bacillus dysenteriæ (Shiga) or allied organisms, Bacillus acidi lactici, Bacillus coli, Bacillus proteus, streptococci, staphylococci, etc., all of which, gaining access to milk, find therein conditions suitable for their growth. No one bacterial type is answerable for all cases, but any milk that contains a large number of any of these organisms may provoke the disorder. The type of disorder known as acute enterocolitis (ileocolitis) may result also from the aforementioned causes, but seems to be due in most cases to Bacillus dysenteriæ, although other organisms, such as the streptococcus, may participate in some of the lesions. This type of affection may occur as a primary disorder, or it may follow acute dyspeptic diarrhœa or gastro-enteritis (cholera infantum); it occurs as a terminal infection in measles, pneumonia, marasmus, etc.,

especially in institutions. Bacillus dysenteriæ is found especially in cases associated with mucus and blood in the stools, and its number is said to be proportionate to the severity of the infection. It is least common in purely fecal discharges, but may be isolated from the intestinal mucosa in fatal cases. Streptococci are often associated with Bacillus dysenteriæ. The mode of infection is not known, although it is attributed to the milk and water. There is some evidence of the contagious nature of some cases. The blood serum may give a positive agglutination test with Bacillus dysenteriæ.

Pathology.—The lesions vary considerably in different cases, but they are usually confined to the colon and the lowermost parts of the ileum. In mild cases there is catarrhal inflammation of the small and the large intestine, with some swelling of the lymphoid follicles; in some cases the swelling of the lymphoid follicles forms a conspicuous feature. Many cases show necrosis and ulceration of the mucosa, sometimes limited to the follicles, sometimes more widespread. In severe cases pseudomembranous inflammation, usually limited to the mucosa, occurs, sometimes irregularly distributed, sometimes widespread—and is associated with submucous œdema and cellular infiltration. The diversity of the pathological lesions found associated with Bacillus dysenteriæ, as well as the large number of associated bacteria, renders it impossible to differentiate the lesions due to the Shiga bacillus from those due to the other intestinal bacteria. In some cases a hyperplastic, followed by an atrophic, condition of the intestinal mucosa ensues and leads to general malnutrition. The mesenteric lymph nodes are swollen, the kidneys are the seat of cloudy swelling, and hypostatic congestion of the lungs and bronchopneumonia occur frequently.

Symptoms.—Of the diarrhœas of children, three types may be distinguished: (1) Acute dyspeptic diarrhœa; (2) gastro-enteritis, gastro-intestinal intoxication, or cholera infantum; and (3) acute enterocolitis.

Acute dyspeptic diarrhœa may commence insidiously or acutely, and may be seen in children of all ages. The attacks may be connected directly with the taking of deleterious food. In many cases, at first, aside from general restlessness and a little looseness and increased frequency of the bowels, the child presents no symptoms. After a day or two the stools increase in frequency, become very malodorous, and contain milk curds and other undigested food (if such has been taken). In other cases the onset is sudden, sometimes with convulsions, but usually with high fever (103° to 104° F.), vomiting, diarrhœa, abdominal pain, tenderness, and distention. The stools vary much in number, and consist of malodorous yellowish or greenish-yellow feces, containing much gas, milk curds, and undigested food. Generally the attack subsides within three or four days (after the intestine has been relieved of irritating contents), but relapses are common, and the lesions may go on to a severe enterocolitis.

Gastro-enteritis, gastro-intestinal intoxication, or *cholera infantum* is an extremely severe though uncommon intoxication (2 to 3 per

cent. of the cases of summer diarrhœa, Holt), that usually develops suddenly in artificially fed children who have been subject to intestinal dyspepsia and diarrhœa. The disease is marked by sudden onset, high fever (105° to 106° F.), protracted vomiting, frequent serous intestinal discharges, marked prostration, frequent small pulse (130 to 180 per minute), early collapse, and frequently early death. If there has been no preceding dyspeptic diarrhœa, the stools are at first copious and frequent, fecal in character, brownish or yellowish in color, and malodorous; soon they become greenish and later grayish, serous, watery, alkaline, and almost devoid of odor, or musty. There may be as many as twenty or thirty stools in the twenty-four hours. The vomiting is often uncontrollable and is augmented by food and drink. At first it consists of the contents of the stomach, highly acid or dense curds; later it becomes watery, mucoid, bile-stained, and less acid or alkaline. The general evidences of intoxication are marked from the beginning: prostration is extreme, the skin is ashen, the features pinched, the eyes and fontanelle sunken, the muscles flabby, the pulse rapid and feeble, the breathing shallow and rapid, the sensorium, at first excited, later becomes benumbed and the child listless; thirst is marked, the urine lessened, the tongue, at first coated and moist, becomes red and dry, and the abdomen retracted. Death may ensue within twenty-four hours in collapse with cold extremities though frequently high internal temperature. With the apathy and coma that precede dissolution, the vomiting and diarrhœa may cease (but should not be incorrectly interpreted). In other cases the severe symptoms abate temporarily or permanently. The patient may remain for several days apathetic, semi-comatose; now and then there may be a convulsive seizure, the head may be retracted, and the respirations irregular, sometimes of the Cheyne-Stokes type (hydrencephaloid, or spurious hydrocephalus), Death may result at the end of three or four days; or the child may go into a speedy, though usually slow, convalescence—a marantic condition (sclerema) persisting for some time; or convalescence may progress slowly or imperfectly and the child ultimately die of marasmus with irregularly recurring attacks of diarrhœa.

Enterocolitis (ileocolitis, follicular enteritis, follicular dysentery), is the form of summer diarrhœa of children commonly associated with Bacillus dysenteriæ, and characterized by necrotic and ulcerative processes in the ileum and the colon. It may follow acute dyspeptic diarrhœa, or any of the acute infectious diseases. The onset is usually gradual, though it may be abrupt. The fever increases and usually remains high (though hyperpyrexia is not as common as in cholera infantum). The stools become very frequent (12 to 30 in twenty-four hours), small in amount, acid in reaction, malodorous, and contain mucus and streaks of blood. Though vomiting may occur, especially at the onset, it is rarely a conspicuous feature of the disease. The abdomen is distended and tender along the course of the ileum. Colicky pains may precede the stools, or the stools may be passed

painlessly, or there may be tenesmus if the lower colon and rectum are involved. In most cases the diarrhœa gradually subsides, the mucus and blood become less, the fever lessens, and the child may be convalescent in two or three weeks. In other cases the convalescence is protracted, the diarrhœa lessens but does not altogether disappear, and the general nutrition of the child suffers, though health may eventually be restored.

In some cases the onset is abrupt and resembles dysentery in the adult. It is more common in elder children (past the third year), and begins with high fever, vomiting, severe abdominal pains, colonic tenderness, marked diarrhœa (frequent small stools containing much mucus and blood), tenesmus, marked intoxication, and great prostration. Death may occur within forty-eight hours. Generally, however, the symptoms abate after twenty-four or thirty-six hours, but the child may continue ill for a week and finally succumb or go into a tedious convalescence.

Under the designation *cœliac disease* has been described, especially by Gee, a variety of intestinal catarrh, of unknown cause, occurring in children from one to five years of age, and characterized by copious, diarrhœic, often frothy, and extremely malodorous stools, resembling oatmeal gruel (diarrhœa alba sive chylosa). The disease begins insidiously, and progresses frequently to a fatal termination, the conspicuous general symptoms being asthenia, anemia, and emaciation.

Diagnosis.—Acute dyspeptic diarrhœa is readily recognized by the fecal character of the stools, which contain undigested food and milk curds. It is well not to attribute any case to teething and similar factors, but to treat it as a disease from the beginning. Cholera infantum is distinguished by the high fever, incessant vomiting, serous discharges, general collapse, and other evidences of severe intoxication. The presence of mucus and blood in the discharges distinguishes enterocolitis.

Prognosis.—The diarrhœas of children are always serious, and the prognosis depends upon the previous condition of the child and the severity of the infection. Acute dyspeptic diarrhœa in a healthy child usually responds to treatment, but may prove fatal in a debilitated child. High fever, incessant vomiting, and extreme prostration are serious signs in cholera infantum.

Treatment.—An essential part of the treatment is prophylaxis. Since most of the cases are related etiologically to the diet, that is, artificial diet, it is important to see that the milk is pure and fresh, that the dairy whence the milk is derived meets all modern requirements, that the milk is kept cool in transit, delivered fresh, and placed again on ice in the household. Pasteurization of fresh milk may be resorted to in hot weather as an additional measure of safety, and all bottles should be sterilized before being used. Additional safeguards consist in reducing somewhat the diet (but not pure water) during the summer, and treating even the slightest symptom of intestinal disorder by stopping

all milk for a day at least, and immediately cleansing the bowel by castor oil or small doses of calomel $\frac{1}{20}$ to $\frac{1}{10}$ grain (0.003 to 0.006 gram) three or four times a day.

Hygienic Treatment.—Hygienic treatment is of the utmost importance, and scarcely anything conduces so much to the comfort and health and recovery of the child as removal from the oppressive air of an overcrowded city to the cooler and more wholesome breezes of the country, mountains, or seaside. When this is impossible, the city child should be kept out of doors in the parks or on the river, and lightly clad. Frequent bathing (tepid water) is an excellent measure, and should be resorted to, especially if the temperature goes above 102° F.

Dietetic Treatment.—The dietetic treatment is of paramount importance. At the onset of the attack of summer diarrhœa, milk must be absolutely prohibited. During the first twelve to twenty-four hours no food should be given, but pure water should be administered in amount equal to that of the fluid food to which the child has been accustomed. In very young and marantic infants (under four months) it may be necessary to resort to food at the end of the first day, as the lesser of two evils, and pasteurized whey or barley water may be given in small amounts. In ordinary cases, however, it is advisable to discontinue milk for at least four or five days, and to substitute barley water (one tablespoonful of barley and a little salt to a pint of water and boil for half an hour, add hot water to make up for loss by evaporation, and strain); or rice water; or albumen-water (the whites of two or three eggs, 1 pint of water, 1 dram of brandy, and a little salt); or beef juice. Older children (three or four years) may be permitted chicken broth, mutton broth, or beef broth, but caution in the use of broths is necessary, since of themselves they are laxative. In some cases the intolerant stomach may be made more retentive by lavage with warm saline solution, and in the event of insufficient water being introduced by the mouth, recourse may be had to enteroclysis. The return to milk diet must be gradual, small amounts of a suitable mixture, well diluted, being added day by day, and being discontinued upon the slightest return of intestinal derangement. In the majority of cases satisfactory milk mixtures may be readily prepared by diluting whole milk one-fourth, one-half, or three-fourths with sterile water or barley water and adding 5 per cent. of lime water and a sufficiency of milk sugar to bring the mixture to the desired percentage. Ordinary whole milk contains: fat, 4 per cent.; sugar, 4.5 per cent.; and proteins, 3.5 per cent. Twenty ounces is a convenient amount of the mixture to make at one time. By adding 5 ounces of whole milk to 1 ounce of lime water, 1 ounce of milk sugar, and enough water to make 20 ounces, a mixture is obtained that approximates: fat, 1 per cent.; sugar, 6 per cent.; and proteins, 0.9 per cent. Ten ounces of milk and the other ingredients, as mentioned, make a mixture approximating: fat, 2 per cent.; sugar, 7 per cent.; and proteins, 1.8 per cent. Fifteen ounces of milk, the lime water and water as mentioned, and $\frac{1}{2}$ ounce of milk sugar make a mixture approximating: fat, 3 per cent.; sugar, 5.5 per cent.

and proteins, 2.7 per cent. In most cases one or the other of these mixtures usually suffices. One should begin with the weaker and proceed to the stronger, remembering that the infants usually can digest fats more easily than proteins. In intractable cases resort should be had to more accurately prepared mixtures. Whey and cream mixtures rather than milk may be found advantageous in many cases for a time at least. A wet-nurse is sometimes advisable.

Medicinal Treatment.—Medicinal treatment is in many cases subsidiary to the foregoing. Since, however, all cases are due to infection of the intestinal contents, and later to infection of the intestine itself, the bowel should be immediately cleared by castor oil, or small and repeated doses of calomel (especially useful in the event of vomiting), or by lavage and colonic irrigation. In acute dyspeptic diarrhœa these measures with regulation of the diet often suffice, though bismuth subnitrate (or subcarbonate, subgallate, salicylate, or beta-naphthol), is useful for its restraining influence on the bowel. In very severe cases, bismuth, salol (or resorcin), and chalk mixture will be found useful; if diarrhœa persists, a little deodorized tincture of opium may be added, but it should not be given until the intestinal tract has been thoroughly cleansed. Pain until then may be relieved by a spice poultice, or spirit of chloroform or camphor.

In cholera infantum, which may be serious and even alarming from the outset, the stomach and intestines should be cleansed with saline solution, or in the event of very high fever, ice water may be used by the bowel, or the child put in a tepid bath, and the water gradually reduced by the addition of ice. An ice-bag should be applied to the head. Morphine, $\frac{1}{100}$ grain (0.0006 gram), and atropine, $\frac{1}{800}$ grain (0.00008 gram), to a child one year old, may be given hypodermicly to allay irritation, and to overcome the effects of the toxemia on the nervous system. It is especially valuable if the vomiting and purging persist and the child collapses. The dose may be repeated in an hour if no improvement has resulted. To replace the lost body fluids, as well as to reduce the toxemia, enteroclysis or hypodermoclysis is of great service. Small amounts, 20 to 40 c.c., repeated, are sometimes more serviceable than a single large dose (250 c.c.). In the collapse of some cases small amounts of caffeine (1 to 400), or of adrenalin chloride, added to the saline solution, may prove useful. Stimulants are indicated in severe cases; sometimes small amounts of brandy, cognac, or champagne may be retained when all else is rejected. Strychnine may be given hypodermicly. When the vomiting has been overcome, bismuth and the remedies previously mentioned, are sometimes efficacious, or starch water and laudanum may be given by the rectum. Return to feeding must be done gradually, beginning with albumin water and beef juice; later whey and cream, peptonized milk, or the milk mixtures mentioned may be given. If convalescence is protracted and diarrhœa persists, or is easily provoked, the diet should be looked to; but the condition may be one of enterocolitis, in which event injections of saline solution

or silver nitrate (1 to 10,000) may be serviceable. Iron, arsenic, cod-liver oil, and change of climate are of marked service during convalescence in many cases. An effective serum for Bacillus dysenteriæ infections has not yet been produced.

CROUPOUS OR DIPHTHERITIC ENTERITIS.

Croupous or diphtheritic enteritis is an inflammation of the intestinal mucosa attended by necrosis and the formation of a croupous (or fibrinous) exudate.

Etiology.—Croupous or diphtheritic enteritis is due to: (1) Bacterial infection—whence it occurs in certain infectious diseases, such as pneumonia (the pneumococcus may be isolated from the exudate in some cases), typhoid fever, scarlet fever, smallpox, and septic processes. (2) It occurs in conditions of reduced vitality, such as occur in chronic diseases, when it is frequently a terminal infection, as in nephritis, cirrhosis of the liver, heart disease, carcinoma, etc.—the reduced vitality permitting of secondary bacterial infection. (3) It occurs also in poisoning by mercury, arsenic, ammonia, lead, etc.

Pathology.—The lesions vary in different cases. In the one there is necrosis of the mucous membrane, varying in extent and depth, but often confined to the tops of the folds of mucous membrane and rather superficial. In other cases the necrosis is more widespread and may involve the entire thickness of the mucosa. The false membrane is grayish, grayish yellow, or grayish white in color, and is surmounted on a congested base. The lesions are more common in the colon than in the small intestine. In the second group of cases the solitary lymph nodes are the seat of swelling and superficial or deep necrosis, attended by the formation of a membranous exudate.

Symptoms.—Often there are no symptoms. When present, the symptoms are those of enteritis and colitis. The exact nature of the disorder cannot be recognized unless some of the exudate is discharged with the stools.

Treatment.—The treatment is that of the primary affection plus that of catarrhal enteritis.

PHLEGMONOUS ENTERITIS.

Phlegmonous enteritis is a rare disease characterized by suppurative inflammation of the mucous and muscular coats of the intestine. As an idiopathic affection it is exceedingly rare (even rarer than phlegmonous gastritis); as a secondary affection it sometimes attends pyococcic processes, and may be associated with intestinal intussusception or strangulation or strangulated hernia. Anatomically the purulent collection may be circumscribed (abscess) or diffuse (phlegmon). The *symptoms* are

usually those of peritonitis, and the affection is scarcely susceptible of differential diagnosis. The *treatment*, apart from operative procedures in association with intestinal obstruction, is unavailing.

APPENDICITIS.

Etiology.—Inflammation of the vermiform appendix is an extraordinarily common disorder: the commonest intra-abdominal disease. It is most common in early adult life, and more common in men than in women. It is really the consequence of infection occurring in an organ provided with a precarious blood supply and ill adapted to drainage—both of which in circumstances permit of the ready operation of bacteria. The factors impeding drainage are the excessive length of the organ, the narrowness and often tortuosity of the lumen, Gerlach's valve, and the swelling of the mucosa that readily occurs. To these one may, perhaps, add the influence of appendicular calculi—which are not fecal concretions in a true sense, but the result of appendicular inflammation; when formed, they tend to keep up the inflammation, and by pressure, attrition, and interfering with local blood supply they are a factor in determining the site of ulceration. Foreign bodies and worms are rarely found in the appendix, but when occurring may be of etiological significance. The chief infectious cause is Bacillus coli, but streptococci, staphylococci, pneumococci, Bacillus influenzæ, Bacillus typhosus, Bacillus dysenteriæ, Bacillus tuberculosis, Streptothrix actinomyces, and other organisms may be the active etiological agent.

Pathology.—Acute and chronic cases may be distinguished. The lesions of the acute cases comprise the ordinary phenomena of inflammation, congestion, œdema, round-cell infiltration, etc., which very rarely may be limited to the mucosa (so-called catarrhal appendicitis). In most cases the lesions, although they vary much in many cases, extend more or less throughout the wall of the appendix. Sometimes they consist only of the aforementioned inflammatory phenomena, with perhaps only a little superficial erosion of the lining mucosa (acute interstitial appendicitis). But in the great majority of cases in which the lesions are widespread or severe there is more or less suppuration, necrosis, and loss of the mucosa (ulcerative appendicitis); the lesions may or may not progress to perforation of the organ. In other cases, in consequence of virulent infection or defective or abolished blood supply, gangrene supervenes. The chronic lesions also are rarely, perhaps, confined to the mucosa (chronic catarrhal appendicitis). In the great majority of cases, however, all the coats of the organ are involved, the conspicuous changes consisting of connective-tissue hyperplasia of the submucosa and the muscularis, round-cell infiltration, and atrophy of the muscular tissue. In some cases, the mucosa having been the seat of more or less suppuration and necrosis, is finally entirely lost, and the opposed ulcerating surfaces by a process of organization may become

united into a firm fibrous union with obliteration of the lumen throughout more or less of the length of the organ (chronic obliterative appendicitis). Most of the chronic cases are subject to acute exacerbations—in which to the chronic hyperplastic process acute manifestations may supervene (recurring or relapsing appendicitis). In many of these cases the appendicular contents become and remain purulent (chronic empyema); and at any time any of the severe acute lesions, such as perforation or gangrene, may supervene.

The chief significance of appendicitis attaches to the peritonitis it provokes. In mild cases this may be strictly localized to the region about the appendix, and serous or serofibrinous in character. In many severe cases, usually with, but often without, perforation, the peritoneal lesions may also remain localized, but they become purulent in character—so-called peri-appendicular abscess. Limiting adhesions may suffice to "wall-off" the abscess from the rest of the peritoneal cavity, but in many cases the pus finds its way into the pelvis, or by a progressive breaking down of adhesions and the formation of new adhesions in advance of the old ones, more or less of the peritoneal cavity may ultimately become involved (progressive fibrinopurulent peritonitis). In some cases there are few, if any, adhesions, the peritoneum constituting a veritable pus sac (diffuse or generalized suppurative peritonitis). Occasionally the lesions become putrid. Rarely the peritoneum is found quite dry and lustreless—in cases of very severe and overwhelming intoxication (toxic peritonitis). The peri-appendicular abscess is sometimes situated behind the peritoneum, about the kidney, near the liver. These extraperitoneal as well as intraperitoneal collections of pus may burrow in various directions—into the intestine, into the pelvis (and be discharged through the rectum or vagina), behind the liver, through the diaphragm, and be expectorated through a bronchus, etc. Multiple abscesses of the liver (suppurative pylephlebitis) is a common complication in neglected cases; general septicopyemia also may ensue. Primary carcinoma is not uncommonly found in appendices removed for appendicitis.

In chronic appendicitis, in addition to the lesions already mentioned, peritoneal adhesions are usually found. These, binding the appendix to adjacent organs, are, in large part, the cause of the symptoms of chronic appendicitis, and may lead to strangulation or kinking of the appendix or intestine and various disturbance of the organs to which they may be attached.

Symptoms.—In *acute appendicitis* the onset is usually sudden. The chief symptoms consist of severe abdominal pain, nausea and vomiting, tenderness and muscular rigidity in the region of the appendix, and fever (101° to 103° F.). The pain is usually sudden in onset and severe, often colicky in character. It is usually at first epigastric or umbilical in situation, or may be diffuse; but it soon becomes localized to the right lower abdominal quadrant. It may radiate to the bladder, to the testicle, or the lumbar region; and may be aggravated by movement of the right thigh, which is

often flexed to relieve the pain. Tenderness also may be more or less general at first, but it soon becomes localized to the region of the appendix, and is usually most marked at McBurney's point (a point midway on a line drawn from the anterior superior spine of the ilium to the umbilicus). In some cases depending upon the direction of the appendix, the tenderness may be most marked in the lumbar region (retroperitoneal appendix pointed upward), in the pelvis, to the left of the median line, etc. The rigidity of the muscles in the right lower abdominal quadrant may be very marked. At the onset, accompanying the fever, the pulse is usually rapid, and a polynuclear leukocytosis commonly supervenes (12,000 to 25,000). The bowels are usually constipated, and painful; and perhaps frequent micturition may occur if the appendix reaches into the pelvis and impinges on the bladder.

In the course of a day or two the lesions and the symptoms may subside and the patient may go on to apparent recovery. Seeming improvement, such as lessening of pain and of the fever, however, by no means indicates subsidence of the inflammatory phenomena; both the pain and fever may lessen in cases attended by perforation and suppuration. The pulse is a much better index of the intra-abdominal conditions than is the pain or the fever. In the cases that do subside the appendix, usually does not return to its previous normal condition, and recurrences and relapses are likely to ensue.

In the cases that do not improve, more or less severe peritoneal lesions develop—either a peri-appendicular abscess or diffuse peritonitis. The peri-appendicular abscess may be recognized by a more or less well-defined mass in the region of the appendix; it is usually doughy to the touch and may disclose deep œdema; it is dull on percussion or perhaps tympanitic from overlying and distended intestinal coils; and on auscultation it reveals no peristaltic sounds. This abscess may follow directly upon what seemed to be a relatively mild appendicitis; that is, there may be no signs whatever suggesting a perforation or other serious complication. Occasionally sudden severe pain does occur, and may be interpreted as rupture of the appendix. Generalized peritonitis is disclosed by generalized pain, tenderness, and rigidity, vomiting, rapid feeble pulse, fever, leukocytosis, Hippocratic countenance, and other evidences of marked intoxication, and final collapse. This generalized peritonitis may be preceded by more or less well-defined evidences of localized abscess, or it may develop directly in consequence of rupture or gangrene of an appendix not enclosed within adhesions.

Chronic appendicitis is characterized clinically by various gastrointestinal and neurasthenic manifestations. The chief symptoms are comprised within the term indigestion (Moynihan's appendix dyspepsia); to which may be added frequent or infrequent attacks of more or less well-marked or frank appendicitis. The dyspeptic phenomena simulate and are readily mistaken for disease of the gall-bladder, gastric ulcer (exaggerated activity of the pylorus, pylorospasm), etc., and often can scarcely be distinguished therefrom aside from operative inter-

vention. Well-marked neurasthenia is quite common and the patient may emaciate. Mucous colitis also may be a manifestation of chronic appendicitis.

Diagnosis.—Sudden severe abdominal pain, nausea and vomiting, tenderness and muscular rigidity in the region of the appendix, and fever, usually suffice for the diagnosis in the acute cases. However, in some cases one has to take into consideration the possibility of the many acute abdominal conditions that may cause sudden pain, nausea, and vomiting, especially perforated gastric or duodenal ulcer, gallstone colic, renal colic, intestinal colic, menstrual pains, acute pancreatitis, Diehl's crisis in floating kidney, intestinal intussusception and strangulation, etc.; but these and other abdominal disorders are in themselves well characterized symptomatically and scarcely any one of them is likely to present all the characteristic symptoms of acute appendicitis. Chronic appendicitis may be recognized in the cases that do not present unequivocal symptoms, in the cases in which dyspepsia is the obtrusive feature, by bearing in mind that most cases of intractable dyspepsia have an anatomical basis, and that this is usually gastric ulcer, gastric carcinoma, perigastric adhesions, chronic cholecystitis, with or without gallstones, chronic pancreatitis, or chronic appendicitis.

Prognosis.—The prognosis in acute cases is extraordinarily uncertain, since no one can foretell in the early stage whether the disease is likely to be mild or severe; the symptoms bear no constant relationship to the severity of the lesions (the most grave anatomical lesions may be present with mild or slight symptoms); and apparent improvement may occur with advancing intra-abdominal conditions. In the event of recovery, relapses and recurrences are likely. The outlook in the chronic cases as to permanent relief is bad unless recourse is had to surgical intervention.

Treatment.—The treatment of appendicitis is surgical; there is no really dependable effective medical treatment. It is true that some patients recover under medical treatment, and it is also true that many die who might be saved by timely resort to surgical measures. Furthermore, it is true that some patients die after an operation, but the great majority of these are subjects in whom the operation has been unduly delayed or they are the subjects of fulminating infections—doomed from the very outset. The mortality following a timely operation is much less than under any other form of treatment. The foregoing are the facts that should guide us in our treatment, and operation should not be delayed until the patient is "sick enough" to warrant it.

Pending the arrival of the surgeon, and in any event as soon as the patient is seen, all food and water should be stopped (and none should be given until the patient is recovering); the stomach should be washed out, and the procedure repeated if nausea and vomiting continue; and the bowels should be opened by an enema. If the patient is seen early, before the development of peritonitis, the bowels may be unloaded by castor oil, but if peritonitis (local as well as general) has supervened, no cathartics should be given by the mouth. An ice bag may be applied

to the abdomen to relieve pain, or hot, moist compresses, if these are more grateful to the patient. Ochsner recommends hot boric-acid solution and alcohol. Enteroclysis by the drop, or the Murphy, method may be used to relieve thirst; perhaps it has also some good effect on the developing peritonitis. If deemed necessary, stimulants and liquid meat extracts (which, however, contain more alcohol than nutriment) may be added to the physiological saline solution, and given three or four times a day. Opium should be entirely withheld if possible; it should never be given until the diagnosis is at least reasonably certain, and then only the smallest possible amount, sufficient only to mitigate the pain and not obtund the sensibilities and mask the symptoms.

SIGMOID DIVERTICULITIS.

(*Perisigmoiditis.*)

Etiology.—Aside from congenital diverticula of the intestine, of which the most common is Meckel's diverticulum, acquired diverticula may occur almost anywhere in the intestine, sometimes in the region of the appendix, but most commonly in the sigmoid flexure of the colon. The cause of the condition is not well understood. They are common in elderly obese subjects who have been constipated. They have been attributed to localized areas of muscular weakness, and are often related to the absorption of the fat of epiploic appendages.

Pathology.—In the sigmoid these diverticula occur either in two parallel rows along the sides of the intestine or along the attachment of the mesentery; they may be confined to the epiploic appendages. There may be several or many. They vary in size from 1 to 2 mm. in diameter to a centimeter or more. When small they are usually spheroidal; but when elongated they are likely to be flask-shaped. They commonly communicate with the lumen of the gut by a constricted orifice. They become the seat of more or less well-marked inflammation, which in acute cases may be catarrhal, suppurative, or gangrenous; and this may give rise to localized peritonitis with abscess formation or to generalized peritonitis—in all of which respects the lesions are somewhat analogous to appendicitis. More or less inspissated fecal matter and even enteroliths may be found within the pouches. In the chronic cases there is usually much connective-tissue hyperplasia and consequent thickening and stenosis of the bowel (chronic hyperplastic sigmoiditis and perisigmoiditis). The condition much resembles and has been mistaken for carcinoma.

Symptoms.—The acute cases most simulate acute appendicitis, the pain, tenderness, and muscular rigidity, however, being on the left side. (As is well known, this may occur in appendicitis, when the appendix reaches over to the left side or depends into the pelvis.) The lesions may go on to abscess formation and divers suppurative complications. In the chronic cases the symptoms consist of constipation, gradually

increasing colonic obstruction, and a tumor mass in the left inguinal region. These occurring in an elder person (usually a man), simulate carcinoma. Doubtless some supposed carcinomas of the sigmoid removed at operation were in reality cases of sigmoid diverticulitis. This sigmoiditis, rather than carcinoma, is suggested by long-standing constipation, gradually increasing intestinal obstruction, absence of blood in the feces, and slight anemia and cachexia.

Prognosis.—The outlook is good under appropriate treatment.

Treatment.—The treatment is wholly surgical and consists of removing the diseased portion of the sigmoid.

ULCERATION OF THE INTESTINE.

Etiology.—The following forms of ulceration of the intestine are described by Nothnagel: (1) Ulceration due to necrotic processes—simple duodenal ulcer, ulcer following burns, embolic and thrombotic ulcers (including those seen in patients with multiple neuritis), and amyloid ulcers. (2) Ulceration due to inflammatory processes—catarrhal ulcer, follicular ulcer, and stercoral or decubital ulcer. (3) Ulceration due to acute infectious diseases—typhoid fever, dysentery, diphtheria, anthrax, sepsis, erysipelas, and varioloid. (4) Ulceration due to chronic infectious diseases—tuberculosis, syphilis, leprosy, and pellagra. (5) Ulceration due to constitutional disease—gout, scurvy, and leukemia. (6) Ulceration due to intoxications—uremic ulcer, mercurial ulcer.

Symptoms.—Duodenal ulceration, as well as typhoid, dysenteric, and diphtheric ulceration, are discussed elsewhere. Diarrhœa with blood, pus, mucus, and shreds of necrotic tissue in the stools are the important symptoms of ulceration of the intestine. Although diarrhœa is common, it is not invariable and may be absent in the presence of even extensive ulceration. Nothnagel believes that the presence or absence of diarrhœa is determined primarily by the site of the ulcerative lesions, stating that if the ulcers are in the small intestine, cecum, or ascending colon they probably never produce diarrhœa unless the ulceration is complicated by other conditions, such as intestinal catarrh, amyloid disease, special forms of infection, or other factors that may influence directly intestinal peristalsis. If the ulcers are in the lower portion of the colon and the rectum, diarrhœa occurs with great regularity, but it may be absent if the ulcers are few in number and if the nervous irritability of these portions of the bowel is lowered or inhibited. Blood is often present, but it may be absent, and if present may be due to causes other than ulceration. It is a valuable sign when it occurs in circumstances that render likely the presence of ulceration. Pus is usually present in greater or less amount, and often requires careful search to detect it; it is of greater diagnostic significance than the presence of blood. Large amounts indicate perforation into the intestine of an extra-intestinal abscess (peri-appendicular, peri-uterine, etc.), the presence of a suppurat-

ing and ulcerating carcinoma, or a croupous inflammation of the intestine. A purulent mucus is sometimes present, and in some cases one may encounter peculiar sago-like grains of mucus, formerly but no longer considered diagnostic of follicular ulceration. Shreds of tissue (mucosa, connective tissue, and even muscularis) are important diagnostically, but they are rather rare. Pain may or may not be present; it is of value only when present continuously at the one place and intensified by pressure. If the ulceration is slight the general nutrition is not much impaired, but extensive ulceration usually leads to marked deterioration of the general health. Secondary infection of the ulcer may cause a little fever, but usually there is none. Perforation is not uncommon, and may give rise to local or generalized peritonitis, or to a retroperitoneal abscess.

Diagnosis.—The diagnosis of the nature of the ulceration may sometimes be made by attention to the attending phenomena and examination of the stools for tubercle bacilli and other specific organisms, as well as shreds of tissue. Tubercle bacilli in the stools, however, may have been swallowed, and do not necessarily mean ulceration of the intestine.

Prognosis.—The prognosis varies with the nature of the etiological factors.

Treatment.—The treatment in many cases is determined by the nature of the primary disorder; the treatment of the primary and the secondary conditions is not separable—such as ulcers occurring in infectious diseases (typhoid fever, dysentery, tuberculosis, syphilis, etc.), in constitutional diseases, in poisoning, etc. In all cases regulation of the diet is of the utmost importance, a suitable diet being determined upon largely by a study of the individual patient. Direct treatment of the ulcers, by means of topical applications or rectal enemas, is scarcely possible unless the ulcers are situated in the rectum or the lower part of the colon, though in some cases even the ascending colon may be reached by enemas. The most suitable medicated enemas consist of boric acid or salicylic acid (0.3 to 0.5 per cent.), thymol (0.1 per cent.), silver nitrate (0.3 to 1 per cent.), or tannic acid (0.3 to 1 per cent.). Recently some good results have been achieved by the operative establishing of an artificial anus through the medium of the cecum and the giving of complete rest to the colon; in other cases, a communication has been established with the exterior through the medium of the cecum or the appendix and the entire colon and the rectum subsequently periodically irrigated with medicated enemas, with asserted good results. Internally few preparations are of much service; but some relief may be afforded the patient by the symptomatic treatment of diarrhœa, pain, hemorrhage, etc.

INTESTINAL OBSTRUCTION.

The term intestinal obstruction is inclusive; it comprises a number of somewhat different though related conditions, to which several names have been applied: Occlusion signifies a complete closure of the lumen

of the intestine entirely arresting the onward passage of the intestinal contents. Stenosis signifies a lessening of the lumen of the intestine, an incomplete occlusion, arising from any cause whatsoever—a condition in which the onward passage of the intestinal contents is impeded, but not arrested. Stricture signifies a lessening of the lumen, the result of disease (usually annular) of the wall of the intestine. Constriction signifies lessening of the lumen of the intestine in consequence of pressure (compression) or traction from without. Obturation signifies obstruction from within the lumen of the intestine, as by impacted feces, an enterolith, a gallstone, etc. Incarceration signifies an imprisonment of the intestine in a part or cavity. Strangulation signifies a form of constriction in which the compression is so severe as to interfere seriously with the vascular and nervous supply, and hence the functional activity and even the life of the intestine. The term ileus is sometimes inappropriately applied to any severe intestinal obstruction or occlusion attended by stercoraceous vomiting; and the term paralytic ileus is sometimes applied to the paresis or paralysis which may follow abdominal operations, the relief of intestinal obstruction (release from constricting bands, rectification of a volvulus, etc.), or that occurs in peritonitis, etc., and sometimes even without anatomical basis.

Etiology.—Intestinal obstruction may be due to:

1. *Disease Outside the Intestine:* (*a*) Tumors of any of the intra-abdominal organs, especially of the ovary, uterus, omentum, or kidney, which give rise to obstruction by compression, the obstruction being usually chronic, though the symptoms may develop acutely. Analogous in action to tumors are abscesses, especially pelvic and peri-appendicular, as well as collections of fluid, such as pancreatic cysts and hydronephrosis. (*b*) Conditions causing strangulation, such as internal hernia (diaphragmatic, subcecal, duodenojejunal, intersigmoid, through the foramen of Winslow); post-inflammatory adhesions and bands, pedicles of tumors, remains of fecal structures, such as Meckel's diverticulum (especially when adherent at its unattached end), fibrous remains of fœtal vessels, etc., beneath which a loop of intestine may slip and become constricted, or which by traction may occasion kinking of the intestine; omental and mesenteric slits and peritoneal pouches, which may engage and strangle a portion of the bowel; etc. Strangulation is the most common cause of acute intestinal obstruction (34 per cent. of the cases, Fitz). Seventy per cent. of the cases occur in men; 40 per cent. between the fifteenth and thirtieth years. In 90 per cent. the small intestine is involved; in 87 per cent. the strangulation is in the right iliac fossa; and in 83 per cent. in the lower half of the abdomen (Fitz).

2. *Disease of the Intestinal Wall:* (*a*) Intussusception or invagination, the infolding of the intestine into itself, is a common cause of acute obstruction (31 per cent. of the cases, Fitz), and occasionally causes chronic obstruction. It is twice as common in males as in females; more than one-half of the cases occur before the tenth year, and many occur in

the first year of life when the intestine is relatively long and irregular; peristalsis (an essential cause of the disorder) is very common. The invagination is always a descending process, the upper portion of the bowel descending into the lower—whence several varieties are recognized: the iliac, in which the ileum alone is involved; the ileocecal, in which the ileocecal valve enters the colon (75 per cent. of the cases); the ileocolic, in which the lower part of the ileum passes through the ileocecal valve; the appendiceal, in which the vermiform appendix enters the cecum; the colic, in which the colon alone is involved; and the colicorectal, in which the colon descends into the rectum. The intussusception forms an irregular tumor varying from less than an inch to a foot or two in length. It consists of three layers—an outermost or receiving layer, a middle or returning layer, and an innermost or entering layer. The intussuscepted intestine is usually red, congested, and much swollen, and about it there is more or less peritonitis. The compression of the mesenteric vessels, especially those supplying the middle and the innermost layers, is often so extreme as to lead to rupture and hemorrhage from the mucous lining and consequent blood in the stools—an important diagnostic feature. Sloughing of the invaginated portion of intestine sometimes results, and should the middle and the outermost layers have become adherent, spontaneous cure may ensue—of which some remarkable instances, with the discharge by the rectum of large portions of the bowel, have been recorded.

(*b*) Volvulus or twist is a fairly common cause of acute intestinal obstruction (about 15 per cent. of the cases). It occurs twice as commonly in males as in females, is most frequent in the fourth decade of life, involves the sigmoid flexure in 50 per cent. of the cases (less commonly the cecum and the small intestine); and is usually axial and associated with a long, non-fatty mesentery. The twist seems sometimes to have been provoked by a trauma or severe diarrhœa.

(*c*) Tumors of the intestine are a rather common cause of chronic obstruction. Cylindrical epithelioma (carcinoma) is the most common. It is usually annular (rarely circumscribed), involves most commonly the sigmoid flexure or the descending colon, and gives rise to a gradual lessening of the lumen of the bowel, until in some cases occlusion is almost if not quite complete. It is more common in males than in females, and occurs especially beyond the middle period of life (except carcinoma of the rectum, which is remarkably common in early adult life). Sarcoma is much less common than carcinoma. Benign growths, such as polyps, adenomas, and fibromas, have been reported.

(*d*) Cicatricial strictures sometimes cause chronic obstruction. The lesions are most common in the large intestine, and follow the healing of dysenteric ulcers (colon), syphilitic ulcers (rectum), follicular ulcers, tuberculous ulcers, and rarely typhoid ulcers. Occasionally the stricture is congenital.

(*e*) Peritonitis, occasioning paralytic distention, is not an uncommon cause of acute or subacute intestinal obstruction.

3. *Abnormal Intestinal Contents:* (*a*) Fecal impaction (coprostasis) is a rather common cause of chronic intestinal obstruction, though the symptoms may develop acutely. The accumulation usually occurs in the cecum or the sigmoid flexure. It may occur at any age, but is most common in children, adult women, and in aged, debilitated, hysterical, hypochondriacal, and insane subjects. The accumulation is sometimes enormous, and may be favored by congenital dilatation of the colon, though habitual inattention to the bowels also may result in atony, distention, and excessive accumulation. The retained fecal masses become very hard, but often permit the passage by them of fluid feces (sometimes interpreted as evidence of the absence of accumulation). Occasionally inflammation and ulceration of the bowel results. (*b*) Gallstones having ulcerated through the gall-bladder into the small intestine sometimes cause obstruction, usually at the ileocecal region, but sometimes in the duodenum. Obstruction is rare if they ulcerate into the colon. (*c*) Enteroliths formed by the deposition of magnesium and calcium salts, especially phosphates, about divers nuclei, such as hard feces, a small gallstone, hair, etc., may cause obstruction. (*d*) Foreign bodies, such as fruit seeds, buttons, coins, etc., as well as large collections of tangled round worms, and occasionally large amounts of bismuth or magnesium taken medicinally, have been known to cause obstruction.

Pathology.—The anatomical results of acute obstruction consist of dilatation of the intestine above the obstruction together with the accumulation of feces and gas, and narrowing of the lumen or collapse of the intestine below the obstruction. In chronic obstruction, above the obstruction there is more or less dilatation of the intestine and hypertrophy of its walls, the latter developing in consequence of the long-standing difficulty to the onward movement of the feces. The intestinal wall, especially the mucous membrane, is often inflamed and ulcerated, and perforation or spontaneous rupture and consequent peritonitis are not unknown.

Symptoms.—Clinically intestinal obstruction may be acute or chronic. Acute obstruction results from strangulation, intussusception, volvulus, foreign bodies, and peritonitis; chronic obstruction results from tumors, cicatricial strictures, fecal impaction, and sometimes intussusception. In many cases, however, in which the obstruction is chronic, that is, in which the narrowing of the lumen develops and progresses slowly, symptoms may for a long time be absent, and ultimately develop suddenly, that is, clinically the obstruction is acute.

Acute intestinal obstruction is characterized by abdominal pain, tenderness, nausea, vomiting, constipation, tympanites, and occasionally a palpable abdominal tumor. Pain is usually an initial symptom, comes on suddenly, is acute and colicky in nature, though after the obstruction is complete it generally becomes continuous, and is always very severe. In the early hours of the attack, at least, it is commonly associated with active (visible and audible) peristalsis. It is not always localized to

the site of the obstruction, but may be referred or diffuse; and it is often associated with abdominal tenderness, which also may be diffuse. Nausea and vomiting follow soon upon the pain, and are sometimes the most distressing symptoms, especially when associated with hiccough, which is not uncommon. The vomit consists at first of the stomach contents (usually food), then (second or third day) of greenish, bile-stained fluid, and ultimately (third, fourth, or fifth day), if the obstruction is complete and the patient is not relieved, of brownish-black, feculent or stercoraceous material—an important diagnostic feature. At the onset of the obstruction, or soon thereafter, the bowels may be opened—a discharge of the feces below the obstruction; but otherwise, in a great majority of cases, constipation is absolute, neither feces nor gas being discharged, though in a few cases the incessant tenesmus may result in the passage of a little mucus, and in intussusception there may be both blood and mucus. Tympanites is a feature of almost all cases, coming on after the lapse of twenty-four hours. It varies much in degree in different cases, being marked in obstruction of the large bowel, but often slight if the small bowel high up is involved. Rarely an abdominal mass may be palpable, especially in intussusception or fecal impaction, but in many cases the exquisite tenderness and pain preclude satisfactory examination. The examination, however, in obscure cases may be facilitated by anesthetization, and a rectal or vaginal exploration may furnish conclusive evidence.

The constitutional symptoms are always severe. The expression is anxious, the face pallid and bathed in cold sweat, the eyes sunken, the pulse rapid and feeble, and soon evidences of collapse supervene. The temperature is usually subnormal, unless peritonitis develops. The urine is diminished, sometimes greatly diminished, and may contain an excess of indican (especially in obstruction of the small intestine), and later some albumin. Leukocytosis, sometimes of high degree, is usually present. If not relieved, the patient usually dies, soon after the third day, with manifestations of peritonitis and auto-intoxication—delirium, coma, muscular twitchings, fever, etc.

Chronic intestinal obstruction varies somewhat symptomatically, depending upon the cause of the obstruction. Cicatricial or cancerous obstruction is characterized by long-standing and increasing constipation, alternating sometimes with attacks of diarrhœa, and frequently associated with colicky pains. The pains, however, often occur at long intervals and are usually attributed to indigestion. The masses of formed fecal matter are sometimes observed to diminish progressively in caliber, but one must remember that fecal masses narrowed by passing through a stricture may acquire ordinary size and contour in the rectum. There is often a sensation of fulness in the abdomen, tympanites develops and is not uncommonly associated with active peristalsis (visible and audible), which often furnishes remarkably trustworthy evidence of how far down the bowel is patulous. Vomiting may occur, but is rather unusual and rarely fecal in character. Attacks of more or less complete, though

temporary, obstruction, due to accumulation of feces above the constriction, may occur from time to time, and are sometimes attended by fever, due doubtless to auto-intoxication. Intestinal hemorrhage is occasionally observed. In some cases a more or less definitely delimitable mass is palpable—usually in the region of the cecum or the sigmoid, but sometimes elsewhere. The patient, as a rule, fails in general health, becomes anemic and cachectic, and ultimately dies from asthenia or, not infrequently, from the consequences of acute obstruction.

In obstruction due to fecal impaction the symptoms, for the most part, are quite similar to the foregoing; constipation is of long standing, but channelling of the fecal masses may permit the discharge of mucus or watery stools. Subjective symptoms are for a long time, in many cases, notoriously slight, despite even great accumulation; but in some cases, abdominal pain and distention may be complained of. When the obstruction is complete the pain and distention are more marked, and vomiting may supervene and even become feculent. Rectal examination, as well as examination along the course of the colon, may disclose scybalous masses, which have a peculiar putty-like consistency and may be somewhat moved about.

Diagnosis.—The diagnosis is concerned with (1) the seat of the obstruction, (2) the nature of the obstruction, and (3) the differentiation from other conditions that may give rise to similar symptoms.

The seat of the obstruction is sometimes impossible of certain determination, but it may be surmised by attention to the age and sex of the patient (having reference to what has already been said in this connection), and by bearing in mind the slightly less frequency, more sudden onset, more acute course, more severe local and general symptoms, more common feculent and protracted vomiting, more likely suppression of urine, excessive indicanuria (due to decomposition of the intestinal contents), and the early development of peritonitis, in obstruction of the small intestine. Obstruction of the small intestine is also suggested by uniform distention of the central portion of the abdomen, with slight or no distention of the lateral portions (situation of the colon), and active, visible, palpable, and audible peristalsis (transverse or oblique ridges). In obstruction of the beginning of the jejunum the distention and peristalsis may be slight and limited to the upper part of the abdomen, or may be altogether absent; but vomiting, gaseous eructations, and hiccough are likely to be marked, and collapse comes on early. In obstruction of the beginning of the duodenum, the symptoms are similar to those of stenosis of the pylorus; in obstruction of the duodenum below the diverticulum of Vater, pancreatic juice may be present in the vomit (but it may be absent in disease of the pancreas, such as carcinoma of the head, which itself may be the cause of the obstruction). Obstruction of the large intestine, if due to strangulation, may quite simulate obstruction of the small intestine, but otherwise it is suggested by slower development of the symptoms of occlusion; less rapid course; later development and less frequency of vomiting which is less likely to be feculent;

limitation of the initial distention and peristalsis to the colon (horse-shoe curve, but frequently irregularly developed); palpable tumor or fecal masses along the course of the colon; more tenesmus with discharge of mucus and blood; less likelihood of anuria; and the later development of peritonitis. A rectal or vaginal examination should not be neglected. In obstruction of the small intestine high up empty and collapsed intestines may be found in the pelvis; in some cases of volvulus of the sigmoid the rectum may be distended (paralytic distention), but contain no feces; and in intussusception the descending intestine may be palpable in the rectum. Some observers have advocated rectal exploration with the entire hand, and if the hand be lady-like this may not be devoid of service. Indefinite or well-defined masses (tumors or fecal masses) may be palpable through the abdominal wall in some cases, even despite considerable tympany, but is much facilitated by anesthetization. Sometimes assistance is obtained by inflating the rectum and the colon with air or distending it with water, the point where the inflation ceases, or the amount of water that may be introduced, suggesting the seat of the obstruction; or after the introduction of air or of bismuth paste a radiographic examination may disclose the seat of obstruction. Sometimes, as suggested by Treves, auscultating while inflating or distending the bowel may afford evidence of the seat of obstruction.

The nature of the obstruction sometimes cannot be diagnosticated, but help may be obtained by attention to the age and sex of the patient, an examination of the palpable openings (for hernia), a rectal or vaginal examination, and the clinical history of the case—acute or sudden onset; an injury to the abdominal wall; previous peritonitis, pelvic or otherwise, localized, or diffuse (suggesting bands or adhesions causing compression or traction); the ingestion of a foreign body; previous gallstone colic; chronic constipation; symptoms of stenosis of the bowel; general failure of health, with the development of anemia, emaciation, and cachexia (suggesting carcinoma), etc. Intussusception is more readily recognized than other conditions: it occurs especially in children, is of sudden onset (sometimes following diarrhœa), is attended by tenesmus and bloody and mucous discharges, and occasionally through the abdominal wall, in the course of the colon (especially the transverse), a peculiar, usually sausage-shaped mass, may be palpable, and may even become hard and firm (contracted) under the hand; in other cases it may be felt through the rectum. Volvulus and strangulation, except in so far as they are suggested by adult life, can rarely be recognized with certainty before operation. Fecal impaction, and in many cases malignant stenosis, should readily be distinguished.

Conditions resembling symptomatically intestinal obstruction are often difficult of differentiation. Internal hernias (obturator, diaphragmatic, internal inguinal ring, etc.) can rarely be distinguished. Other conditions which may occasion confusion are acute appendicitis, acute pancreatitis, acute peritonitis, acute enteritis (especially in children), intestinal colic, gallstone colic, nephritic colic, torsion of the pedicle

of an ovarian or uterine tumor or of a floating kidney, a phantom tumor, etc. In many cases of acute obstruction peritonitis develops so promptly as to be really part and parcel of the disease. In these, as well as in other cases, the diagnosis cannot go beyond acute obstruction.

Prognosis.—The prognosis varies, depending upon the nature of the obstruction, and in many cases upon the promptness of appropriate treatment. In acute obstruction the prognosis is always grave, a fatal termination occurring usually in from three to seven days unless the condition is relieved. In chronic obstruction symptoms are often present for months, until, as in carcinoma, the patient eventually dies of asthenia, or rather suddenly from acute obstruction. When due to fecal impaction recovery usually follows clearing of the bowel. In the event of certain complications, such as gangrene, perforation, peritonitis, etc., the outlook is practically hopeless.

Treatment.—A knowledge of the pathological conditions present in intestinal obstruction suggests the futility of medicinal treatment in all but the exceptional case, and in this the fortunate outcome is often the result rather of good luck than of good management. Excepting only fecal impaction, intestinal obstruction is essentially a surgical disease and should be treated on surgical principles. Since in many cases it is impossible to differentiate between the different forms of intestinal obstruction, and since delay only adds to the danger, operation should be undertaken with the least possible delay.

Pending the advent of the surgeon, the stomach should be washed out repeatedly with warm saline solution—a mode of treatment warmly advocated by Kussmaul and others, and tending to relieve not only the persistent vomiting, but also the intestinal distention and pressure above the obstruction, and thus mitigate the violent peristalsis and the pain. Irrigation of the rectum and colon is also of much service in some cases and may occasionally, especially in intussusception in children, result in the reduction of the intussusception—though it is not wise to expect this as a necessary result of the treatment. The amount of fluid injected should be measured with a view to determine the distance of the obstruction from the anus. Under no circumstances whatever should purgatives be administered; even in fecal impaction they should not be given until, after repeated and copious oil or watery enemas, the major portion of the impacted feces has been discharged.

The question of the use of opium before operation has been much debated. Personally, I prefer not to use it, if it can be avoided. In some cases, however, the pain is really intolerable, and if it cannot be relieved by hot or cold applications externally and atropine internally, morphine may be given in small dose—the smaller the better, since in the event of operation its deleterious effects may become only too apparent. Recently a number of good results have been reported from the use of atropine in very large doses, $\frac{1}{32}$ to $\frac{1}{12}$ grain (0.002 to 0.005 gram) twice or thrice in the first twenty-four hours; the pain has been relieved and spontaneous recovery has resulted. The treatment merits further trial.

That some patients have withstood repeated and even forcible distention of the rectum and colon, drastic cathartics, violent shakings, strenuous kneading of the abdomen, etc., merely illustrates the wonderful resources of the human economy; and that some patients have succumbed to this treatment (production of perforation, etc.) rather than to the disease, as such, cannot well be doubted. Personally, I should prefer the chances of operation, in what is admittedly a very serious disease, to the hazard and hope of medicinal treatment.

DILATATION OF THE COLON.

Dilatation of the colon may be due: (1) Gaseous accumulation, which, though usually transitory, is of significance from the fact that from pressure upward it may seriously embarass the action of the heart and lungs, particularly if these organs are diseased. (2) Fecal accumulations, with which gallstones, enteroliths, and foreign bodies may be associated. (3) Obstruction of the colon, usually carcinomatous or a volvulus; less commonly cicatricial stricture, sigmoid diverticulitis, etc. (4) Idiopathic dilatation of the colon (Hirschsprung's disease; Virchow's giant growth of the colon). This is usually congenital and is seen in children in more than three-fourths of the cases. There may be stricture of the sigmoid, but usually there is no apparent obstruction. The colon may attain really a giant growth, as in Formad's case, in which it measured 15 to 30 inches in circumference, and with the contained feces weighed 47 pounds. The chief *symptoms* are obstinate and prolonged constipation, alternating, perhaps, with periods of diarrhea, markedly protuberant abdomen, especially in the course of the colon, and in some cases visible colonic peristalsis. The disorder is intractable, auto-intoxication, emaciation, in some cases complete intestinal obstruction, peritonitis, etc., eventually ensuing. *Treatment* is unsatisfactory. Treves has successfully removed the greater part of the colon.

INFARCTION OF THE MESENTERIC VESSELS.

Embolism of the mesenteric arteries (most commonly the superior mesenteric artery) may occur in arteriosclerosis, vegetative endocarditis, aneurysm, etc. Thrombosis of the mesenteric veins may follow directly on infections, especially ulcerative process in the intestine (appendicitis, intestinal ulceration), or it may develop secondarily in cirrhosis of the liver, pylephlebitis, and anemic and cachectic states; and it usually follows arterial embolism. Gangrene of the supplied part of the intestine follows; if the area blocked is small, an efficient collateral circulation may be established, but if the superior mesenteric artery (virtually an end-artery) is involved, death is inevitable. The *symptoms* are usually abrupt in onset, and consist of sudden abdominal pain, nausea, vomiting, and bloody diarrhœa. Soon peritonitis sets in, and the patient usually

succumbs within two or three days. In a few cases the onset is more insidious and the symptoms not necessarily obtrusively intestinal. The *diagnosis* is rarely made with certainty. It may be suspected in the event of endocarditis or aneurysm, which may furnish the obstructing embolus. Usually the diagnosis is intestinal obstruction. Operation and resection of the gangrenous bowel is occasionally successful; otherwise treatment is unavailing.

INTESTINAL SAND.

Of conditions described as intestinal sand there are two—the true and the false. True intestinal sand (intestinal lithiasis) consists of small, usually dark-colored, gritty particles formed of an organic base, but made up largely of inorganic salts (often 75 per cent. or more), especially calcium phosphate and carbonate. It is more frequent in women (two-thirds of the cases) than in men, especially in nervous and hypochondriacal subjects, between the thirtieth and the fortieth year.

Etiology.—The process of the formation of *true intestinal sand* has been likened to that of appendicular calculi and gallstones. Duckworth and Garrod believe that since the sand contains only traces of unaltered bile pigment and no cholesterin (distinction from gallstones) and is rich in urobilin, it must be formed in the upper part of the colon, where the conversion of bile pigment into urobilin is well advanced, and which is structurally better adapted than the small intestine for the lengthy sojourn necessary for the deposition of earthy salts. *False intestinal sand* is made up of the residue of certain articles of food and occasionally of medicines. The sclerenchymatous particles of pears (pear sand), particles of bananas, seeds of fruits (figs and different berries), fish bones, grain residues, etc., have been observed as forming so-called intestinal sand. Olive oil, in consequence of partial digestion, has been known to form masses resembling gallstones (whence the erroneous opinion as to the efficacy of the olive oil treatment), and substances like salol may also give rise to so-called (false) intestinal sand.

Symptoms.—Clinically the discharge of the sand is usually attended by more or less, sometimes very severe, colicky pain, due to constipation or to mucous colic (with which it is often associated). Clinically, as a rule, no pain attends the discharge of false intestinal sand, any pain complained of being likely due to constipation, for the relief of which, figs, etc., may have been taken.

Treatment.—The treatment is that of the associated conditions, such as intestinal colic, constipation, and mucous colic.

HEMORRHOIDS.

(*Piles; Hemorrhoidal Phlebectasis.*)

Etiology.—Hemorrhoids, or varicose dilatations of the inferior hemorrhoidal veins, is a purely local disease, due to mechanical obstruction of the

circulation, and is caused by: (1) Local disease of or about the rectum, such as fecal accumulation, rectal inflammations, strictures and tumors of the rectum or adjacent structures (uterus, ovary, bladder, prostate, etc.); (2) obstruction to the portal circulation due to cirrhosis and other disorders of the liver and portal system; and (3) chronic passive congestion due to heart disease. Sedentary occupations, repeated pregnancies, straining at stool, etc., are believed to act as contributing factors.

Pathology.—Hemorrhoids may be external or internal, or both. Internal hemorrhoids are in close relation with the sphincter, but sometimes they extend high up in the rectum, and even into the sigmoid. External hemorrhoids may consist of (*a*) a small venous tumor, that develops suddenly (in consequence of rupture of one of the external hemorrhoidal veins), is dark purplish in color, and very tender; (*b*) small tabs of skin and connective tissue, which originally contained some more or less dilated and tortuous veins. They may be single or multiple, pedunculated or sessile, and not infrequently surround the anus. They rarely occasion symptoms. Internal hemorrhoids may be (*a*) diffuse, that is, thickened, dilated, tortuous, and closely aggregated venous cords and capillaries (sometimes also arterioles); usually granular on inspection, and covered with a very thin and readily torn mucous covering—whence the frequent bleeding (bleeding piles); (*b*) circumscribed tumor masses, single or multiple, roundish or irregular in outline, and more or less encircling the bowel. They may be pedunculated or have a broad base of attachment; they are bright red when inflamed, and dark reddish or purplish when much congested or filled with clot (due to rupture of a vessel). They may become extruded, and in consequence partially or completely strangulated by the sphincter, or ulcerated, and give rise to more or less hemorrhage.

Symptoms.—Small and even moderately large hemorrhoids often exist without occasioning any symptoms, though examination may reveal their presence. Symptoms, when present, vary with the number, size, and condition of the hemorrhoids. Probably the commonest complaints are sensations of discomfort, sometimes a more or less constant desire to evacuate the bowel, a temporary dissatisfied sensation following defecation, and anal itching and burning. Defecation is often painful, sometimes so painful as of itself to engender voluntary constipation. The final evacuation of the hardened feces only induces more severe pain, and eroding the piles provokes more or less abundant hemorrhage. When the piles are inflamed, ulcerated, or strangulated, blood and mucus may pass from the bowel altogether independently of the stools. Sometimes a high grade of secondary anemia is induced; hypochondriasis and melancholia are by no means uncommon.

Diagnosis.—External piles are readily recognized by inspection; internal piles necessitate digital exploration or protoscopic examination for their recognition.

Prognosis.—Since the causes of hemorrhoids are so largely chronic, their course is usually progressive. In themselves they are not fatal,

except in so far as neglect may lead to serious and even fatal complications, such as strangulation and gangrene.

Treatment.—The treatment is either palliative or curative. The curative treatment is surgical—ligation or excision—and may be resorted to in the great majority of cases. Many patients, however, will not submit to operation, and an operation may be ill advised in subjects with advanced heart or liver disease. Marked benefit may usually be secured in many cases by securing a daily evacuation of the bowels, by habits of regularity, attention to the diet, and saline aperients if necessary. Local cleansing is of the utmost importance. Applications of ice water or of an ice bag, or even a cold sitz bath after the daily evacuation, is of benefit. Local pain and itching may be relieved by belladonna, ichthyol, or iodoform (by suppository), by the fluid extract of hamamelis, or by equal parts of gall and belladonna ointment. In other cases excellent results follow the use of iron subsulphate, 2 to 3 grains (0.13 to 0.20 gram) by suppository, or a 10 to 15 per cent. ointment. Intractable hemorrhage, ulceration, strangulation, etc., call for surgical intervention.

VISCEROPTOSIS.

(*Splanchnoptosis; Enteroptosis; Gastroptosis; Coloptosis; Hepatoptosis; Nephroptosis; Splenoptosis; Glenard's Disease*).

Visceroptosis is an inclusive term used to describe the condition in which the abdominal viscera fall from their normal to a lower position in the abdomen. The stomach, intestine, transverse colon, and the kidneys are most commonly involved, but any or all of the abdominal viscera may "drop" in the individual case.

Etiology.—Two causative factors are operative—a congenital weakness or laxity of the mesentery and ligament supporting the abdominal organs, and lack of the normal support afforded by the abdominal wall. The majority of cases occur in women, in whom, in consequence of repeated pregnancies, ill-fitting and tightly laced corsets, etc., the abdominal wall becomes lax and flaccid. In both sexes factors like traumatism, excessive muscular strain, such as may be associated with chronic constipation and severe coughing, emaciation, recurring ascites, etc., may lead to the disorder. In some, especially young subjects, there seems to be undoubtedly a congenital weakness of the supporting ligament and mesentery, since there is often no undue laxness of the abdominal wall.

Pathology.—Visceroptosis in itself is, perhaps, not to be looked upon as a disease *sui generis;* aside from their abnormal position, the organs may be quite normal.

Symptoms.—Several classes of cases may be encountered. In the one, even considerable grades of ptoses may exist without notable symptoms. In a second series of cases, obviously due to a markedly relaxed abdomen (repeated pregnancies), there may be more or less marked symptoms.

In a third series, the conspicuous symptoms are neurasthenic (nervousness, headache, backache, asthenia, anemia, emaciation, etc.), with special complaints directed to the gastro-intestinal tract. These comprise, as a rule, the more common manifestations of indigestion, such as poor or capricious appetite, epigastric pain and distress, gaseous and acid eructations, flatulence, nausea, and constipation; in some cases there is hyperacidity, in others motor insufficiency. Examination reveals the stomach dropped, *in toto*, the lower border two or three inches or more below the umbilicus, and the upper border correspondingly ptosed: a depression in the epigastrium and a fulness about the umbilicus. The stomach is usually more vertical than normally, especially in women who have laced tightly; and it may also be dilated. Waves of peristalsis may be visible, but they are much weaker than those due to organic obstruction; occasionally, however, considerable pyloric obstruction may be caused by malposition of the pylorus, perigastric adhesions, a displaced right kidney, etc. The intestines, also, especially the colon, may be much displaced. The transverse colon may fall as low as the brim of the pelvis, occasioning a so-called V- or M-shaped colon; and the sigmoid may be markedly redundant: in both cases constipation is usually a marked symptom.

Ptosis of one or both kidneys, usually the right, is often associated with gastro-enteroptosis, but it may occur independently thereof. There is usually more or less pain in the lumbar region, or a sense of weight and dragging; it may be so severe as to simulate and be indistinguishable from renal colic; or it may be a part of Dietl's crises: severe pain, chills, fever, nausea, vomiting, and collapse; the urine is usually lessened in amount and may contain blood and pus, and the kidney may be enlarged and tender. The cause of these crises is not well understood; they have been attributed to twists or kinks of the renal vessels or ureters, obstruction of the bowels, etc. On examination, the kidney, when ptosed, may be just *palpable* on deep inspiration, which is quite common, especially in women and on the right side, and is not of much consequence; it may be so *movable* that on deep inspiration it descends to about the level of the umbilicus, and may be retained there by the palpating hand during expiration; or it may be *floating*, quite low, and readily pushed about to or beyond the median line, in the iliac fossa, etc.

Ptosis of the liver and spleen is much less common, and may be readily recognized by physical exploration.

Diagnosis.—The diagnosis is readily made upon physical examination. The most trustworthy results follow radiographic examination, in the case of the stomach and intestine, after inflation with air or bismuth paste. Some diagnostic importance has been ascribed by Stiller to a floating tenth rib, which he believes the most common stigma of the basic congenital asthenic condition; but a floating tenth rib occurs under other circumstances.

Prognosis.—The outlook as regards relief of symptoms and improvement in the general health is good. One must remember that even con-

siderable degrees of ptosis may be present without notable symptoms. So long as the motor power of the stomach, for instance, is efficient, symptoms may be entirely in abeyance.

Treatment.—The underlying neurasthenic condition demands the most careful attention—hydrotherapy, regulated feeding, general tonics (especially nux vomica), change of scene and air, etc. Gastric hyperacidity or subacidity, flatulence, and constipation must be treated, as mentioned under these headings. Much relief may be afforded by a properly made, fitted, and applied bandage or abdominal supporter; in fact, the bandage in itself may lead to symptomatic cure. Occasionally resort may be had to operative measures—to elevate a markedly displaced stomach or colon, to resect a redundant sigmoid, etc.

DISEASES OF THE LIVER.

Pathological Physiology.—The functions of the liver are: To detoxify poisons that enter the body by way of, or are elaborated within, the gastro-intestinal tract, or are perhaps produced elsewhere; to secrete bile; to warehouse some of the excess of fat taken as food and to release it when the supply from without becomes deficient; to warehouse glycogen derived from the carbohydrates taken as food, or from the non-nitrogenous part of the proteins when the supply of carbohydrates is deficient, and to convert the glycogen into glucose and liberate it as it is required by the system; and to assist in the metabolism of the proteins, to the extent, at least, of forming urea from ammonia compounds. There are other functions, such as erythrocytolysis, etc., that are of minor interest in the present connection.

Disorder of the functions of the liver may be primary or secondary; and it may be partial, involving perhaps only one function, or it may be total, involving presumably all the functions. The belief in a primary general disorder of function is well exemplified in the terms torpid liver, biliousness, bilious headache, lithæmia, etc. Present-day opinions tend, perhaps unwisely, to minimize the influence of the liver in these disorders; but this seems an almost inevitable result of the knowledge that phenomena commonly attributed to a supposed torpid liver and the other affections mentioned may result from factors that do not involve the liver primarily, if at all, and that uric acid (lithemia), by no means the important factor in disease it was once thought, is not formed exclusively in the liver. The dyspeptic symptoms of a supposed torpid liver are usually due to a gastro-intestinal catarrh set up by dietetic indiscretions, excessive eating (especially of proteins and carbohydrates), the consumption of too much alcohol, etc.; the spread of this catarrh to the diverticulum of Vater and the common bile duct, or perhaps rarely a toxic descending radicular cholangitis, is answerable for the subicteric tint of the conjunctiva and skin common in these subjects; and the headache and other nervous symptoms result from the absorption and circu-

lation in the blood stream of enterogenic toxins. The liver usually participates in the process only secondarily; perhaps the enterogenic toxins are produced in such amounts that the liver is incapable of neutralizing them and they pass over into the circulation, or they are so virulent that they impair the functional activity of or set up congestive and other alterations in the liver. In either event the symptoms are gastro-intestinal rather than hepatic in origin, and the liver, responding to excessive demands and being in consequence actively congested, is often, for a time at least, of increased rather than of decreased functional activity. The bearing of these facts on treatment is obvious and material.

There is, however, another series of cases, such as the passive congestion of chronic cardiac and pulmonary disease, in which there is reason to believe that the functional activity of the liver is reduced. It is not unlikely that the headache and other nervous symptoms, digestive disturbances, and loss of flesh and strength that occur in these cases are due, in part at least, to the disordered functions of the liver. This is perhaps due directly to the influence of enterogenic poisons which, being carried to the liver by the portal circulation, encounter an organ reduced in vitality by the long-continued passive congestion, and incapable of exerting as it should its detoxifying function; the toxins, in consequence, pass over into the general circulation and cause at least some of the symptoms.

In another series of cases, more or less complete functional failure of the liver ensues. These are cases of severe intoxication or infection, in which widespread destruction or disorganization of the hepatic parenchyma occurs. The striking clinical phenomena, which may develop suddenly, although they are sometimes preceded by minor symptoms, are for the most part nervous; they are variously designated hepatargia, acholia, cholemia, hepatic auto-intoxication, etc. Headache is a conspicuous symptom, and is soon followed by mental excitement, delirium (which may be very active or maniacal), muscular twitchings, and convulsions; or mental hebetude progressing to deep coma may ensue. Most of these cases are associated with varying grades of jaundice and other phenomena that will be more fully discussed under jaundice and acute yellow atrophy of the liver.

Disturbances in the detoxifying function of the liver is believed by some observers to be the basis of the eclamptic manifestations of uremia, pregnancy, etc. Disorder of the fat metabolism often leads to acidosis and acid intoxication, due to the presence of acetone, diacetic acid, and β-oxybutyric acid in the blood. These substances are formed principally in the liver and the muscles, but probably also in other tissues. They result from imperfect oxidation and are directly related to deficient glycogen in the liver and muscles—which may be the consequence of insufficient intake (as in starvation), or of excessive output (as in diabetes).

Alimentary levulosuria has been suggested as a test of the functional

capacity of the liver, and is perhaps of some diagnostic significance in cirrhosis. The test should be performed as follows: Food should be withheld after the evening meal until the test has been completed. In the morning the patient should void his urine, and then (upon an empty stomach) he should take 100 grams of levulose dissolved in 500 c.c. of water. Weak tea without cream may be substituted for the water if preferred by the patient. At the end of an hour, and hourly thereafter for four hours, the patient should void his urine. The specimens should be examined separately for levulose, preferably by the polariscope, or by the Seliwanoff method, which is as follows: Equal parts of the urine and of a half-and-half solution of concentrated hydrochloric acid and water should be mixed in a test-tube, and a few grains of resorcin added. The mixture should then be boiled, whereupon, if levulose is present, a beautiful red color appears; this deepens on standing and a dark-red deposit settles in the test-tube. If the disorder of the liver be of moderate severity, the levulose will be found in the third or fourth specimen of urine, perhaps in the fourth only; if there be profound alteration of the hepatic structure, the levulose may be found in the first specimen of urine, as well as in the subsequent specimens, and it may persist for several additional hours.

JAUNDICE.

Jaundice is a term applied to staining of the tissues with bile pigments —bilirubin and biliverdin. It is merely a symptom, and is recognized clinically by a yellowish or yellowish-green discoloration of the skin and visible mucous membranes and by the presence of bile pigment in the urine.

Types of Jaundice.—Jaundice can result only from: (1) An obstruction somewhere in the course of the biliary tract, in consequence of which the free flow of the bile being impeded, it becomes absorbed by the lymphatics or bloodvessels—jaundice from stasis of bile; or (2) disturbances in the functions of the liver cells, whereby the bile is diverted from the biliary capillaries to the lymphatics or the blood channels—jaundice from parapedesis of bile. In the one case the disorder involves the bile after it has been formed and entered the biliary channels; in the other it involves the bile pigments while they are still within the hepatic cells.

Obstructive or Mechanical Jaundice.—In the event of obstruction to the extrahepatic or intrahepatic biliary ducts the outflow of bile is hindered, while the hepatic cells still continue to secrete bile, so that the biliary ducts, even to the finest biliary radicles (capillaries), become often enormously dilated. Finally, the biliary capillaries rupture into the lymph spaces between the hepatic cells and the bloodvessels; thence by way of the lymphatic vessels and the thoracic duct the biliary constituents reach the blood stream and the tissues of the body. This type of jaundice may be found in cases of: (1) Obstruction within the common bile duct or the hepatic duct; such as gallstones, parasites, foreign bodies,

etc.; (2) inflammatory, cicatricial, or neoplastic stenosis of the ducts; (3) compression of the ducts from without by tumors of the pancreas, stomach, intestine, gall-bladder, regional lymph nodes, kidney, retroperitoneum, mesentery, etc., inflammatory adhesions, swollen lymph nodes, fecal accumulations, aneurysm of the aorta or of the hepatic or mesenteric artery, etc.; (4) kinking or torsion of the ducts the consequence of gastroptosis, hepatoptosis, nephroptosis, the pregnant uterus, tumors of the abdominal or pelvic organs, etc; and (5) diseases of the liver unattended by gross obstruction of the extrahepatic biliary ducts, such as localized inflammatory processes (abscesses), tubercles, gummas, hydatid cysts, primary and secondary newgrowths within the substance of the liver, etc.

Hemolytic, Toxic, Infectious, Hemohepatogenous Jaundice.—Cases of jaundice in which no obstruction of the biliary ducts can be found were formerly described as non-obstructive or hematogenous, in contradistinction to the obstructive or hepatogenous cases. But Naunyn, Minkowski, Stadelmann, and others definitely proved that all jaundice is hepatogenous—in the sense that bile pigments are formed only in the liver. Since, however, in many cases increased destruction of erythrocytes provides the material (hemoglobin) from which the bile pigments are formed in excess (polychromia), these cases are sometimes spoken of as hemohepatogenous jaundice; and since they occur in toxic, infectious, and other conditions associated with increased hemolysis, they are spoken of as hemolytic, toxic, or infectious jaundice.

The mechanism of this type of jaundice is not well understood. Frerichs believed that the bile found its way directly into the bloodvessels, in consequence of changes in the tension of the contents of the hepatic cells and of the bloodvessels—either an increase in the intercellular pressure or a decrease in the portal pressure. Liebermeister believed that the disordered liver cells lost their ability to retain or fix the bile, and that in consequence it became diffused into the bloodvessels or lymphatics—so-called diffusion or akathektic jaundice. Minkowski assumed a disturbance in the normal currents within the liver cells, so that the bile became diverted from the biliary capillaries to the bloodvessels or lymphatics—jaundice from parapedisis (paracholia of Pick). It is probable that the toxic and infectious causes of this form of jaundice seriously compromise the functional integrity of the liver cells; but it has also been demonstrated that they provoke also a radicular cholangitis—which obstructs the free flow of bile. Eppinger states that in these cases thrombi made up of coagulated bile occur in and obstruct the biliary capillaries; in consequence thereof, mechanical stasis of the bile develops in the finest biliary capillaries, and, as in cases of gross obstruction of the larger extrahepatic ducts, the biliary canaliculi dilate and ultimately rupture into the pericellular lymph spaces. In these cases there is at first an increased formation and excretion of bile (polycholia) and of bile pigments (polychromia, pleiochromia); soon, however, the excretion of bile lessens, the bile that is excreted becomes viscid, and

finally excretion may cease—which may be attributed to a radicular cholangitis or the biliary thrombi, or both. Thus, although this form of jaundice is properly called toxic, infectious, or hemolytic, it is at the same time hepatogenous and obstructive.

This hemolytic jaundice occurs in many infections, such as the different types of so-called infectious jaundice, syphilis, yellow fever, septicopyemia, malaria, pneumonia, typhoid fever, etc.; in intoxications, such as poisoning with ptomaines, phosphorus, arseniuretted hydrogen, chloroform, mushrooms, toluylenediamin, pyrogallol, snake venom, coal-tar products, etc.; in acute yellow atrophy of the liver; occasionally in cirrhosis and other diffuse diseases of the liver; in progressive pernicious anemia and hemoglobinemia; in disturbances of the circulation, such as passive congestion; in certain nervous perturbations (so-called emotional jaundice, menstrual jaundice, both of more than doubtful occurrence); in the newborn; and as a chronic family or hereditary disorder.

In a number of cases, in addition to the jaundice, there are phenomena of general infection, such as fever, neuromuscular pains, enlargement of the spleen and liver, gastro-intestinal disorders, such as vomiting and diarrhœa, nervous manifestations, such as headache, stupor, etc., often leukocytosis, albuminuria, tube casts, etc.; to these cases the term infectious jaundice (Weil's disease) has been applied. The not uncommon epidemic occurrence of such cases warrants the use of the term *epidemic jaundice*. In other cases the clinical phenomena are more severe, symptoms attributable to hepatic insufficiency (hepatargia), to poisoning by bile acids (cholemia), or to hepatic auto-intoxication (acidosis), supervene, and a fatal issue usually ensues; the condition is spoken of as grave jaundice (*icterus gravis*). When, in addition, considerable destruction of the liver cells occurs, the term acute yellow atrophy of the liver is employed. No sharp line of distinction can be drawn between these cases; cases mild in the beginning may progress to the most severe and fatal (acute yellow atrophy); and the degree of jaundice bears no constant relationship to the demonstrable changes in the liver, nor to the severity of the other clinical phenomena.

Icterus neonatorum is a peculiar form of jaundice that develops in from two-thirds to three-fourths of newborn infants on the second or third day of life, and usually fades within a week or ten days. The cutaneous discoloration is slight, and is commonly unattended by other symptoms. The urine is often of normal color, and usually does not contain soluble bile pigments, but may reveal biliary granules or crystals in desquamated renal epithelium. The nature of this form of jaundice is not well understood. It has been attributed to compression of the biliary capillaries by distended portal radicles; to catarrhal cholangitis (Virchow); and to biliary stasis in consequence of the diminutive size of the bile ducts and consequent obstruction (Kehrer and Cohnheim). Frank attributed it to absorption of bilirubin from the meconium; and Quincke has suggested that this bilirubin, which is not converted into urobilin on account of the absence of intestinal fermentation in the newborn, being absorbed,

reaches the general circulation by way of the ductus venosus without traversing the liver. Most likely it is really due to the erythrocytolysis of the early days of life; that is, it is hemolytic in nature. This benign form of jaundice must be distinguished from a more severe and fatal form that may also occur in the early days of life in consequence of congenital syphilis, septicopyemia, Winckel's disease, congenital obliteration of the bile ducts, etc.

Chronic family jaundice (hereditary jaundice, congenital hemolytic jaundice, acholuric jaundice, splenomegalic jaundice, hemolytic icteroanemia) is a form of chronic non-obstructive jaundice (first accurately described by Minkowski) that is usually hereditary or occurs in several members of the same family, although some cases are "acquired." It may date from birth or develop during adolescence; and it usually lasts for years without notable impairment of the general health. Its chief characteristics are slight or moderate jaundice, enlargement of the spleen, well-colored stools, the presence of urobilin and urobilinogen in the urine (but no bile pigments), moderate anemia, and occasional attacks of fever, of biliousness (following dietetic indiscretions), and of epigastric pain, etc. (due to complicating gallstones). Itching, slow pulse, multiple hemorrhages, xanthelasma, and other usual accompaniments of jaundice are commonly absent. The disease is due to increased fragility of the erythrocytes, or their decreased resistance to hemolyzing agents (Chauffard). In the differential diagnosis one has sometimes to take into consideration all the aforementioned types of jaundice occurring soon after birth, as well as (in older subjects) Hanot's biliary cirrhosis—which occasionally occurs in several members of the same family.

Effects of Jaundice.—All of the tissues and organs, with the exception of the nervous, are stained with biliary pigments; and in some severe cases of toxic or infectious jaundice the nervous system also may be stained. The staining is due, in part, to the deposition of biliary pigments in the tissues, in part to the circulation of blood and lymph containing undeposited pigments. It is most marked in the skin, mucous membranes, serous membranes (endocardium, intima of vessels, etc.), the liver, kidneys, heart, sweat, serous transudates, exudates, etc. The saliva, tears, mucus, and milk are not stained unless an inflammatory exudate be added to them.

Jaundice is manifested first by a yellowish discoloration of the conjunctivæ; in mild cases the discoloration is demonstrable there only. The skin becomes of a characteristic lemon-yellow, orange-yellow, greenish-yellow, olive, or olive-bronze color. The discoloration is light in many cases of toxic or hemolytic jaundice, in which it is rarely more than moderate in grade; it is light also in mild cases of catarrhal cholangitis; it is darkest in cases of permanent and complete obstruction of the common bile duct, and it may become so dark—olive bronze or greenish black—as to warrant the designation black jaundice (melanicterus, icterus melas). The intensity of the jaundice in these cases is proportionate to the completeness of the obstruction of the common

duct, or, in case the obstruction involve one of the larger intrahepatic ducts, to the size of the duct implicated. Pruritus is common and distressing; rarely it precedes the discoloration of the skin. Urticaria and considerable sweating (often localized to the palms) are sometimes observed. Local infections, such as boils, occasionally occur. In chronic cases, local telangiectases of the skin (sometimes also of the oral and other mucous membranes) tend to develop, and flat or nodular yellowish, chamois-leather-colored patches (xanthelasma, xanthoma, vitiligoidea) may appear on the skin or mucous membranes.

Bradycardia is a characteristic feature, especially of acute cases and those of short duration. The pulse rate may fall to 60, to 40, even to 21 (Frerichs) a minute, and it is subject to sudden fluctuations upon slight provocation. This has been attributed to a local inhibitory action of the bile acids on the cardiac muscle or ganglia; but recent investigations suggest that perhaps the action is on the central nervous system.

Bile pigments are present in excess in the circulating blood, but whether they exert any deleterious effect on the blood itself has not been satisfactorily determined. The clotting time of the blood is much increased —from the normal three to four minutes to ten to twelve minutes—a fact of much importance when surgical operations in jaundiced patients are under consideration. In protracted and severe cases there is a marked tendency to subcutaneous, submucous, and subserous hemorrhages.

In jaundice a considerable portion of the biliary derivatives is excreted in the urine, which, in consequence, is discolored yellowish, yellowish red, greenish, or greenish-brown, depending upon the relative amounts of bilirubin, biliverdin (its oxidation product), or urobilin (its reduction product). These bile pigments may be found in the urine several hours, sometimes even several days, before there is any obvious discoloration of the skin or visible mucous membranes; they constitute, thus, the first clinical manifestation of jaundice. In addition, the urine often contains a number of hyaline, sometimes bile-stained, casts—attributable to irritation of the convoluted and Henle's tubules provoked by excreting the bile pigments (which are not excreted by the glomeruli). In other and particularly long-standing cases, in addition to casts, the urine is diminished in amount, of increased specific gravity, and contains more or less albumin—an expression of nephritis due probably to associated toxic conditions rather than to the jaundice *per se*. Urobilin and urobilinogen are commonly found in the urine. They are formed in the intestine by the action of bacteria on bilirubin; they are absorbed and carried to the liver by the portal circulation. Some diagnostic significance has been attached to their presence, as well as that of phenol, in the urine in disease of the liver. The occurrence of urobilinuria in cases of jaundice, however, does not justify the designation "urobilin jaundice," since the cutaneous pigmentation is due to bilirubin and biliverdin.

The functions of the liver, in addition to the formation of bile, may be disturbed—reduction in the glycogen content and lessening of the detoxifying function. The liver cells are sometimes much degenerated, not

always, however, directly in consequence of the jaundice. Eppinger has shown that in these cases marked regenerative changes occur to replace cells destroyed. In advanced cases of obstructive jaundice pericholangitic fibrosis sometimes occurs, and has been described as a form of biliary cirrhosis. The absence of bile from the intestinal tract results in the passage of large, pale, grayish, slate-colored or clay-colored, pasty, fetid acid stools. The pale color is due, in part only, to the absence of bile; in part it is due to the large amount of imperfectly digested fats, which may be increased from the normal 7 to 10 per cent. to as much as 80 per cent. Microscopically and chemically the fats are found to consist of free fatty-acid crystals and soaps (calcium and magnesium) of the higher fatty acids; neutral fats are present in very small amounts. Rarely pale or decolorized stools contain biliary derivatives in considerable amounts—determinable by chemical examination; this occurs especially in disease of the pancreas attended by obstruction to the outflow of the pancreatic juice, but unassociated with jaundice. The pale color of the feces in these cases is due to the large amount of fat, and to the reduction of bilirubin to leukourobilin. Constipation is the rule, but sometimes the bowel movements are frequent, perhaps diarrhœic, which may be due to large fecal masses resulting from imperfect digestion of fat and to the laxative action of the free fatty acids.

Grave disturbances of the nervous system occur in many cases of jaundice; on the one hand, there are manifestations of depression, such as general neuromuscular weakness, asthenia, headache, vertigo, mental depression (that may progress to melancholia), insomnia, etc.; on the other hand, there are manifestations of irritation, such as severe headache, active delirium, coma, convulsions, etc. These manifestations are more common in cases of acute toxic or infectious jaundice (associated with fever) than in the more protracted cases of obstructive jaundice; but they may supervene in any case of jaundice and not uncommonly lead to the fatal issue. They are commonly described under the name cholemia, but it is difficult to separate from one another the symptoms due to bile acidemia, to interference with the detoxifying function of the liver, and to the initiating toxic or infectious process. The condition is not cholemia in a restricted sense—that is, it is not due to bile acidemia, since the symptoms may occur when bile acids are not in excess in the blood, as well as in cases of disease of the liver (such as cirrhosis) unassociated with jaundice. The condition is most likely an acid intoxication, due to disturbances of the detoxifying function of the liver, which may be brought about by disorganization of the hepatic parenchyma. Xanthopsia and hemeralopia also are probably due to a toxic disturbance of the retina, rather than to the sometimes assumed discoloration of the media of the eye.

Man is able to withstand the effects of jaundice for a long time. The fatalities that ensue in the acute, as well as in the chronic, cases are due not so much to the jaundice per se as to other causes—the underlying cause of the jaundice, acid intoxication, etc. In long-standing cases, however,

more or less disturbance of the general nutrition gradually ensues—due largely to imperfect fat absorption. To supply sufficient calories to prevent breaking down of the body tissues, there must be considerable increase in the protein intake; this results in loss of appetite, disordered digestion, perhaps diarrhœa, etc., so that, the intake being reduced, the patient must utilize his own fat or protein tissue to cover the continued loss, and, having no means of restoring the deficit, a very dangerous inanition results.

ANOMALIES OF FORM AND POSITION.

Congenital Anomalies of Form.—The liver, in consequence of defects of development, is occasionally found in the thoracic cavity or projecting beneath the skin near the umbilicus (hepatomphalos); in monsters the organ may be entirely absent. In partial and complete transposition of the viscera the liver may be as much in the left half of the body as normally it is in the right; in this event the left lobe is the larger.

Acquired Anomalies of Form (Deformities).—The important acquired anomalies of form consist of (1) the so-called corset or constricted liver; and (2) linguiform lobulation. *The corset* or *constricted liver* is the consequence of tight lacing, and is therefore almost wholly confined to the female sex (as much as 25 per cent. of female cadavers in some series); but an analogous condition is sometimes encountered in men, the result of the constricting influence of a tight belt or strap. The basis of the deformity is a pressure atrophy of the liver parenchyma followed by fibrosis. Sometimes there is only a deep circumferential furrow; usually the liver is considerably elongated and markedly thinned at the point of greatest constriction (most marked atrophy), which often corresponds with the plane of the upper pole of the right kidney. Various grades of this condition are encountered, and it gradually merges into that spoken of as *linguiform* (or *Riedel's*) lobe (partial hepatoptosis). This also is common in female subjects, but it is intimately related to cholelithiasis and cholecystitis, enlargement of the gall-bladder being found in 60 per cent. of the cases. These linguiform lobes are often long and much attenuated; frequently they are attached to the main portion of the liver by a thin fibrous band; and they are often extremely mobile (partial hepatoptosis). The corset liver and a linguiform lobe may be entirely symptomless. In some cases there are symptoms of indigestion, as likely due to the tight lacing *per se* as to the changes in the liver. A lingual lobe is sometimes painful and tender, due to congestion or associated cholecystitis and cholelithiasis; occasionally nausea and vomiting ensue, and have been attributed to interference with the functions of the pylorus and duodenum; occasionally pains of biliary colic occur. In other cases the abnormality of the right lobe is detected accidentally, and may be mistaken for an abdominal tumor.

Treatment.—The corset liver is better prevented than cured. The symptoms attributable to a linguiform lobe are most amenable to the

surgical measures appropriate to the cure of cholecystitis and cholelithiasis, to which they are largely due.

Acquired Anomalies of Position.—The liver may be displaced upward, downward, or laterally, or it may be rotated on its anteroposterior axis (anteverted or retroverted), usually the result of disease of adjacent thoracic or abdominal organs.

The important acquired anomaly of position is that variously spoken of as hepatoptosis, wandering liver, movable liver, etc., a condition in which the liver is abnormally movable and presents variously in the abdominal cavity. It is mentioned in connection with visceroptosis (page 685).

DISEASES OF THE BLOODVESSELS OF THE LIVER.

Hepatic Artery.—*Dilatation* is observed in cirrhosis and other conditions when the portal vein is obstructed. *Thrombosis* is very rare. *Embolism* occasionally occurs; it may be mechanical, infectious (provoking multiple abscesses), or sarcomatous (usually melanotic from the eye). *Aneurysm* is rare (40 cases). The chief symptoms are pain, jaundice, and hemorrhage into the alimentary tract.

Hepatic Veins.—*Dilatation* occurs in chronic heart disease attended by failure of the right ventricle. *Thrombosis* is rare, but may occur in various sorts of disease in and about the vein. The lesions are analogous to those of chronic passive congestion. It may be suspected in cases of aggravated passive hyperemia upon the sudden onset of ascites. *Embolism* (*retrograde embolism*) occasionally occurs from reversal of the blood current, due to vigorous contraction of the right ventricle, coughing, sneezing, and other violent expiratory efforts, and the forces of gravity. The lesions vary with the nature of the embolus (mechanical, infectious, neoplastic). *Suppurative phlebitis* may occur in pyogenic infections of the liver. *Chronic obliterating hepatic endophlebitis* is rare. The condition resembles and is usually mistaken for cirrhosis.

Portal Vein.—*Dilatation* is part of portal cirrhosis, and is attributable to increased blood pressure. *Thrombosis* (*pylethrombosis, pylephlebitis adhesiva*) is rather common in cirrhosis of the liver and in intra-abdominal newgrowths, but it may occur also in inflammatory disorders of the abdominal viscera. The thrombosis may begin in the portal radicles within the liver, in the main trunk of the vein, or in the peripheral radicles anywhere within the area of collection of the vein; and subsequently, by extension, the process may become widespread. In some cases the disorder seems to be due to a low-grade infection. The portal vein may become converted into a fibrous cord (pylephlebitis adhesiva), and a collateral circulation may become established. The *diagnosis* is rarely possible, but is suggested by sudden onset of ascites of high grade, gastro-intestinal hemorrhage, enlargement of the spleen, abdominal pain, and symptoms of intestinal obstruction. Death may occur soon; at all events, usually within two years. The *treatment* is palliative; the ascites

may require repeated tapping. *Embolism* of the intrahepatic branches of the portal vein is a common sequence of inflammatory, suppurative, ulcerative, and neoplastic processes in the organs within the area of collection of the portal vein. *Suppurative pylephlebitis* is common, especially in early adult life, and, like adhesive pylephlebitis, may begin in the portal radicles within the liver, in the main trunk of the vein, or in the peripheral radicles anywhere within the area of collection of the vein; and subsequently, by extension, the process may become more or less widespread. Usually the disorder is secondary to ulcerative or suppurative processes of the gastro-intestinal tract, especially appendicitis, dysenteric and other ulcerations of the intestine, etc. The infecting microörganisms are staphylococci, streptococci, Bacillus coli, Bacillus dysenteriæ, etc. The *symptoms* consist of those of the antecedent disorder, to which are gradually added (if they have not already existed) irregular chills, fever, sweats, polynuclear leukocytosis, and prostration; as well as extension of abdominal pain beyond the seat of original disease, enlargement and tenderness of the liver, perhaps also of the spleen, and sometimes vomiting, hematemesis, and diarrhœa. Usually the patient soon passes into a typhoid state and dies. *Chronic portal endophlebitis* rarely occurs: as a primary disease (syphilis, splenic anemia with sequential Banti's disease, etc.), or as a secondary process (chronic passive congestion and cirrhosis of the liver). Partial or complete thrombosis may follow.

FATTY LIVER.

Fatty liver is a comprehensive term applicable to the different conditions characterized by an excess of fat in the liver. It includes fatty infiltration, in which there is an excessive deposit of fat in otherwise unaltered or slightly altered liver cells; and fatty degeneration, in which, in addition to the fat, there are greater or less degenerative changes in the hepatic parenchyma, perhaps the direct conversion of the liver cells into fat. Formerly the two conditions were differentiated; doubtless there are extreme cases of fatty infiltration and of fatty degeneration, but many cases partake of the nature of both processes, and, contrary to former opinions, there is now considerable trustworthy evidence to suggest at least that the fat in the liver under all circumstances is derived mainly from adipose tissue elsewhere in the body.

Etiology.—The causes of fatty liver are: (1) Dietetic indiscretions and the disorders of metabolism that tend to produce obesity, of which fatty liver (mostly infiltration) is a conspicuous feature; (2) anemic and cachectic conditions, such as occur in the late stages of tuberculosis, amyloid disease, carcinoma, etc.; (3) passive congestion; (4) poisons, such as alcoholism, phosphorus, arsenic, antimony, chloroform, etc; (5) certain local infections of the intestinal tract, such as the gastro-intestinal catarrh of children, dysentery, etc.; (6) certain general infections, notably streptococcic, staphylococcic, and other pyococcic infections of long duration (prolonged suppuration), tuberculosis, etc.

Pathology.—Usually the liver is enlarged, and may weigh 3000 to 4000 grams; it is of lessened specific gravity (sometimes floating in water), and of lessened resistance; it is normal in conformation, and has a smooth surface and rounded edges, and is pale yellowish in color. Histologically, fatty infiltration and degeneration may be combined in varying proportions.

Symptoms.—The symptoms of fatty liver are ill defined. In general, the symptoms are those of the primary disorder-obesity, tuberculosis, alcoholism, toxemic conditions, etc. In perhaps the majority of cases there are no symptoms referable directly to the liver; occasionally sensations of weight may be due to the mere weight of the liver, and ill-defined dyspeptic symptoms may be attributed to functional inactivity of the liver, although they are more likely to be due to associated disorders of the stomach and intestines, improper eating and drinking, etc. The liver is enlarged and commonly palpable two or three fingers' breadth below the costal margin.

In *phosphorus poisoning* the early symptoms are those of gastro-intestinal irritation (epigastric distress, nausea, and vomiting). If the patient does not die soon, there is commonly a period of quiescence, that to the unwary suggests recovery and speedy cure. At the end of two or three days, however, vomiting and epigastric distress return, jaundice supervenes, the pulse becomes weak and accelerated, the heart dilated, subcutaneous, submucous, and subserous hemorrhage develop, and the patient usually dies within a week. In most cases the liver is enlarged throughout, smooth and tender; in some cases it decreases in size before death.

Diagnosis.—Minor grades of fatty liver cannot be recognized with certainty; the more marked grades must be differentiated from other causes of enlargement, such as the fatty cirrhotic liver, amyloid liver, leukemic infiltration, passive congestion, displaced liver, etc.

Prognosis.—The prognosis depends upon the primary cause.

Treatment.—The treatment is altogether that of the primary disorder.

ACUTE YELLOW ATROPHY OF THE LIVER.

Acute yellow atrophy of the liver is an acute, widespread autolytic necrosis of the liver cells, characterized clinically by jaundice, reduction in the size of the liver, and toxic disturbances of cerebration, proceeding to a fatal issue.

Etiology.—Acute yellow atrophy of the liver is especially frequent between the twentieth and thirtieth year (50 per cent. of the cases), and in women (two-thirds of the cases). Both of these facts are explainable largely by the frequent occurrence of the disease during pregnancy; almost if not quite one-half of the cases in women occur about the middle or during the latter half of pregnancy. The causative influence of pregnancy is akin to that exerted by the toxemia of pregnancy, in which the

liver frequently shows degenerative changes, and in the production of which it plays an important, if not the important, part. Various known infections exert some etiological influence; thus 10 per cent. of the cases occur during secondary syphilis (in which slight jaundice is not very rare); pyogenic infections (septicopyemia, osteomyelitis, erysipelas, etc.), typhoid fever, malaria, diphtheria, etc., may antedate the liver disorder. Etiological significance is also attributed to certain non-bacterial poisons, such as alcohol, ptomaines, chloroform, phosphorus, etc. Chloroform is of undoubted significance, being responsible for the cases of so-called delayed chloroform poisoning. Phosphorus gives rise to a somewhat analogous condition, but the lesions in the liver are not alike in the two disorders. Similar phenomena may develop in the course of other diseases of the liver, such as advanced passive congestion, cirrhosis, cholangitis, etc. All of the foregoing leads to the conclusion that the designation acute yellow atrophy, as well as icterus gravis, comprises a series of diverse disorders that exhibit more or less superficial resemblance. We are now able to separate some of the cases from the main group, such as those due to syphilis, septicopyemia, puerperal eclampsia, phosphorus poisoning, delayed chloroform poisoning, etc., but the definite etiological factor of the major group is still unknown.

Pathology.—The liver is much reduced in size and weight, being often one-half or one-third that of the normal (800 to 500 grams or less). The organ is flaccid and has so lost its normal elasticity and resistance that it may be folded or bent upon itself. On sectioning the liver it is found to be dense and resistant; the section surface is mottled—yellowish or yellowish-red areas of varying size and configuration alternating and more or less gradually merging the one into the other. The yellowish coloration is due chiefly, if not entirely, to bilirubin rather than to fatty alterations (the fat content is usually not increased, and may be diminished); the reddish areas seem to be an advanced stage of the yellow, and to be more numerous or larger the longer the patient lives. Examination of the fresh liver tissue (scrapings of the section surface) reveals necrotic liver cells, erythrocytes, leukocytes, leucin, tyrosin, and xanthin. Histologically there is intense and widespread (not focal) destruction of the liver cells. Ultimately the cells disappear, apparently in consequence of autolysis, and leave behind the supporting framework of the liver, granular debris, and endothelium of the capillaries (the reddish areas on macroscopic inspection). The small bile ducts become obstructed in consequence of desquamation of their lining epithelium and participate in the production of the jaundice. In some, especially protracted, cases there are conspicuous evidences of attempts at regeneration (compensatory hyperplasia) of the liver cells; pseudobiliary canaliculi derived from the liver cells as well as the smaller bile ducts also are often conspicuous. All the organs are more or less bile-stained. The spleen is soft, and is slightly or moderately enlarged in about two-thirds of the cases. The kidneys are enlarged, softened, pale, opaque, and often reveal small foci of hemorrhage, cloudy swelling, and fatty changes.

Small hemorrhages are found beneath the skin and the various serous membranes (pleura, pericardium, peritoneum, meninges) and mucous membranes (gastro-intestinal, respiratory, and genito-urinary tract), and are doubtless attributable to toxic changes in the blood and blood-vessels.

F. W. White supports the view, originally advanced by Flexner, that the process is autolytic in nature, and that it is brought about by some poison having a specific action on the liver cells whose life it destroys without injuring the proteolytic ferments they contain, whence autodigestion ensues. The leucin and tyrosin result partly from autolysis of the liver, and partly from non-conversion into urea of leucin and tyrosin formed elsewhere. Other evidence of this autolysis is found in the albuminose (decomposing protein) and purin bodies (destruction of nuclein) sometimes encountered in the urine. The reduction in the excretion of urea and the increased excretion of ammonia are interpretable as efforts on the part of nature to neutralize increased acidosis, whence the process may be looked upon as an acid intoxication. White believes that the chief symptoms of the severe intoxication are due largely to the damage done to the functions of the liver, and that the special poisonous substances are probably: (1) Nitrogenous antecedents of urea; (2) certain products of decomposition in the bowel which are normally deprived of their toxic power in passing through the liver; and (3) decomposition products of the destroyed liver tissue; and, further, that this view is borne out by observation of other severe diseases of the liver in which little or no jaundice occurs.

Symptoms.—The initial symptoms are those of gastro-intestinal catarrh, soon (one to four days) followed by jaundice; these may last a variable period, usually five to seven days, but sometimes several weeks. During this period the disorder is usually looked upon as ordinary catarrhal jaundice (cholangitis), and as a matter of fact there is little if anything to suggest the more serious disorder. The patient complains of malaise, perhaps fugitive neuromuscular pains, poor appetite, coated tongue, occasional vomiting, constipation, etc.; there is usually moderate jaundice and bile pigment in the urine. Soon, however, the graver symptoms supervene. The jaundice deepens, becoming dark yellowish or bronze-like, slightly greenish (in rare, extremely rapid cases jaundice may be absent); severe nausea and vomiting set in and are often associated with hematemesis; diarrhœa may supervene; and grave nervous symptoms ensue —headache, mental confusion, hebetude, and restlessness, soon followed by a noisy delirium, and later coma and perhaps convulsions. Hemorrhages (disordered nutrition of the vessel walls and hemolytic action of bile salts on the erythrocytes) occur beneath the skin and into and from divers mucous membranes (intestines, urinary tract, respiratory tract, genital tract, retina, and most commonly the stomach); abortion may occur in pregnant women. Soon the patient passes into a profound typhoid state. There may be slight fever (pyococcic infection), but usually the temperature is below normal (until shortly before death, when there may

be an agonal rise); the pulse becomes rapid, feeble, and of low tension; the respirations become accelerated and irregular; the pupils dilate (said to be important, diagnostically); the tongue becomes dry, furred, and tremulous; sordes collect on the teeth and gums; and there are fibrillary twitchings of the muscles, etc.

The liver in some cases is found slightly or moderately enlarged in the early days of the disorder; this may be due to preëxisting disease (as cirrhosis), but rarely it seems to be a part of the acute yellow atrophy itself. Soon, however, the liver dulness diminishes rapidly in extent, until it may be entirely absent or constitute a small area in the axillary region. Slight or moderate enlargement of the spleen may be made out in about two-thirds of the cases.

The feces, as a rule, are dark and offensive. The dark color in the early stages is that of normal feces; that of the feces late in the disease is probably due, in part at least, to contained blood, which obscures the paucity of the bile, since very little if any bile probably enters the intestine in the advanced stages of most cases. The stools may be light colored, especially if there be diarrhœa.

The urine, as a rule, is lessened in amount, of increased specific gravity, of increased acidity, and high colored (bile pigment and excess of urobilin); it usually contains a small amount of albumin, and hyaline, granular, fatty, and epithelial tube casts. The highly significant changes consist in the presence of leucin, tyrosin, and the other amino-acids (largely the result of autodigestion of the liver); sarcolactic, diacetic, and other fatty acids (lactic, acetic, butyric, and succinic); marked reduction in the amount of urea with an associated increase in the amount of ammonia and other nitrogenous substances. A moderate leukocytosis (up to 16,000) may be present. Occasionally there may be ascites.

Diagnosis.—Careful studies of the urine, especially in pregnant women, and in cases of mild jaundice, and the detection of significant urinary changes, especially evidences of an acid intoxication, should enable us to recognize the antecedent conditions and perhaps ward off the more serious later stages. The development of nervous symptoms in any case of jaundice should always awaken suspicion. When fully developed, jaundice, vomiting, delirium (and other cerebral symptoms), lessening in the size of the liver, and the presence of leucin and tyrosin in the urine are unmistakable (although leucin and tyrosin may be found in other conditions).

Phosphorus poisoning may be excluded by the absence of a history of the ingestion of phosphorus (not always trustworthy), the latter development of jaundice (which in phosphorus poisoning usually supervenes after a day or two of symptoms referable to disordered stomach), decrease in the size of the liver (which usually remains enlarged in phosphorus poisoning), the more severe nervous symptoms, and the constant occurrence and larger amounts of leucin and tyrosin. The liver of phosphorus poisoning may contain 30 per cent. of fat, as contrasted with 5 per cent. in acute atrophy.

Prognosis.—The disease is almost invariably fatal. A few cases of recovery have been reported.

Treatment.—In the early stages the patient should be confined to bed. Milk is the best food, and should be diluted with some alkaline mineral water; cereals and other carbohydrates may be given in considerable amounts, since they are likely to prevent or limit the acid intoxication. The bowels should be opened freely and repeatedly, with a view to remove all noxious matters from the intestine, as well as material from which toxic substances may be formed; calomel and the saline cathartics seem the best. Insistence should be laid upon the drinking of large amounts of an alkaline water. Alkaline diuretics and other alkalies (sodium bicarbonate) and enteroclysis and hypodermoclysis may be resorted to. Intestinal antiseptics are indicated theoretically. Stimulants, such as alcohol, strychnine, and digitalis, are useful for the general vital depression and collapse; an ice-cap to the head, the bromides internally, and a warm bath or sponge, or a small amount of morphine ($\frac{1}{32}$ to $\frac{1}{10}$ grain, 9.002 to 0.006 gram) or of cocaine ($\frac{1}{8}$ grain,0.008 gram) hypodermically, may control the restlessness and delirium; warm applications to the abdomen, and cocaine, dilute hydrocyanic acid, bismuth, and carbolic acid, or small amounts of champagne, etc., may control the vomiting.

ACTIVE CONGESTION OF THE LIVER.

Etiology.—Active congestion of the liver, usually toxic in nature and gastro-intestinal in origin, may result from: (1) The processes of digestion, when it is a normal condition; (2) endogenous and exogenous poisons that may be carried by the portal vein (alcohol, overeating, ptomaines, bacterial toxins, etc.); (3) bacterial toxins that may reach the liver by way of the general circulation (typhoid fever, malaria, pyococcic infections, etc.); (4) "cold," "a chill upon the liver," the "tropical liver;" and (5) perhaps vasomotor disturbances (menstruation, etc.). Most of these factors are the causes also of inflammation and degenerative changes in the liver; it is difficult, if not impossible, to separate the one set of conditions from the other; they are often combined.

Symptoms.—The cases associated with dietetic indiscretions are manifest by so-called indigestion, or the complex popularly designated "biliousness" (poor appetite, coated tongue, epigastric distress, nausea, occasionally vomiting, constipation, headache, etc.). Perhaps there is a subicteric hue to the scleræ and the skin; the liver may be found slightly enlarged and tender. In the cases met in infections, such as dysentery, malaria, etc., the constitutional symptoms are more marked; slight or moderate fever is usually present (progression to acute hepatitis); and the local gastro-intestinal symptoms may be more or less in abeyance, although they may become aggravated during digestion. The liver becomes enlarged and tender.

Diagnosis.—The diagnosis is usually apparent from the etiological factors, the general disturbances of the gastro-intestinal tract, the slight

jaundice, and enlargement and tenderness of the liver; by no means of minor diagnostic importance is recurrence of attacks in a person of known ill-chosen habits of eating and drinking. The diagnostic difficulties consist in determining whether or not to the congestion permanent damage to the liver tissue has been added. With recurrence of attacks, their increasing duration, and lessening intervals, this becomes the more likely. In the more acute cases encountered in infections, aggravation of the constitutional symptoms, together with enlargement and tenderness of the liver, usually suffice for diagnosis. In these cases there are usually parenchymatous changes in addition to the congestion.

Prognosis.—The disorder rarely lasts more than three or four days; sometimes a week. It is not dangerous in itself, but acquires its importance because of its etiological factors (especially alcohol) and its frequent recurrence in the indiscrete: in those that persist in the use of alcohol, in whom it is likely to eventuate in cirrhosis; and in those continuously exposed to other etiological factors, such as malaria, dysentery, etc., in the tropics, in whom freedom from recurrences can only be obtained by removal to temperate zones (freedom from etiological causes).

Treatment.—The treatment is that of acute non-suppurative hepatitis.

PASSIVE CONGESTION OF THE LIVER.

Etiology.—Passive congestion of the liver, largely mechanical in nature and cardiac in origin (whence, cardiac liver), occurs: (1) In acute and chronic failure of the heart, the ultimate condition in myocardial, pericardial, and chronic valvular disease; (2) in advanced stages of such diseases of the lungs as cause increased work of the right side of the heart and finally lead to its failure (chronic bronchitis, emphysema, chronic adhesive pleuritis, etc.); and (3) from local factors that obstruct the circulation about the junction of the hepatic veins and the inferior vena cava (kyphosis, inflammatory adhesions, angulation by a displaced heart, pleural effusion, etc.).

Pathology.—Three stages of passive congestion of the liver may be distinguished: (1) The merely congested or engorged liver, found in cases of acute or recent heart failure (common in infections, such as pneumonia, typhoid fever, etc.), and in chronic valvular disease, with well-maintained compensation, in which death has occurred suddenly or after a few days' illness only; (2) the congested nutmeg liver, found in cases of long-standing heart disease, with dilatation of the right heart and tricuspid insufficiency, in which compensation, although precariously maintained, has been on the whole fairly satisfactory, and in which no serious or prolonged failure has occurred until that which leads to death; and (3) the fibrotic nutmeg liver (red atrophy of the liver), found also in cases of long-standing heart disease with dilatation of the right heart and tricuspid insufficiency, in which one or more severe and pro-

longed attacks of failure of compensation have occurred, during which likely the auricle has suffered especially, and, being perhaps totally incapacitated, has permitted the full force of the ventricle to be transmitted to the hepatic lobules with consequent rupture of the venules and capillaries, laceration and destruction of hepatic parenchyma, and its repair by newgrown connective tissue (scar tissue). It is likely that congestion of the liver in the early stages of cardiac failure (whether due to endocardial, myocardial, pericardial, or pulmonary disorder) is a compensatory process; that this is especially active in acute failure, such as occurs in many infections and in the early stage of chronic valvular disease; that not until the liver has been distended to and beyond its capacity does œdema ensue; and that with the progress of th valvular defect and the gradual loss of the safety-valve action of the liver œdema appears more and more easily.

Symptoms.—The symptoms consist of those due to the primary cardiac or pulmonary disease, especially dyspnœa, cyanosis, and œdema, and those due more directly to the disorder of the liver itself. These comprise especially pain and distress or a sense of weight and heaviness in the right hypochondrium, and gastro-intestinal phenomena, such as poor appetite, epigastric distress especially after eating, flatulence, nausea, and constipation. These are due in part to general venous engorgement, in part to portal congestion transmitted from the congested hepatic veins. Slight or moderate jaundice is common; the admixture of the jaundice and more or less cyanosis gives to the patient a very characteristic bluish or purplish, green or yellow discoloration. Œdema, followed by ascites, is often present, being part of the cardiac decompensation. Occasionally the ascites is disproportionately large in amount and may require repeated tappings; in this event it is associated with the fibrotic congested liver or with chronic adhesive peritonitis (perihepatitis). The liver is enlarged, perhaps reaching to or below the transumbilical line; it is tender, and it may reveal an expansile ventricular systolic pulsation.

Diagnosis.—The diagnosis is usually evident from a knowledge of the primary disorder, an enlarged and tender liver, and other evidences of failing cardiac compensation.

Prognosis.—The prognosis is altogether that of the primary disorder.

Treatment.—The treatment is that of the primary cardiac or pulmonary disorder; virtually it is that of failing cardiac compensation.

PERIHEPATITIS.

Acute Perihepatitis.—Acute perihepatitis is almost if not always a secondary disorder, due to antecedent disease of the liver or adjacent viscera. The lesions may be localized or diffuse, and fibrinous, serofibrinous, or purulent in character. The fibrinous exudation may undergo organization and lead to filamentous or dense adhesions; the suppurative lesions may be part of one form of subphrenic abscess.

The important *symptoms* consist of local pain and audible and palpable frictions. There may be a short, hacking cough (perhaps set up by manipulation of the liver), hiccough (suggesting involvement of the diaphragm), and nausea and vomiting. The *diagnosis* is often obscured by the predominance of the symptoms of the primary disorder. The *treatment* is that of the primary disease and local measures (strapping, counterirritation, etc.) to relieve the pain. Operation is required in the suppurative cases.

Local Chronic Perihepatitis.—Local chronic perihepatitis usually follows and is therefore due to the same causes as the acute process. The lesions are usually quite firm adhesions that serve to bind the neighboring organs variously to one another. The symptoms are scarcely, if ever, definite, but local pain, tenderness, and rigidity may suggest the disorder.

General chronic perihepatitis will be discussed under diseases of the peritoneum (multiple serositis, page 750).

ACUTE HEPATITIS.

Acute Non-suppurative Hepatitis.—The term acute non-suppurative or parenchymatous hepatitis is employed to include a series of most diverse disorders that exhibit all stages of progression from acute congestion to the inflammatory focal necroses of many infections, and the more widespread process manifested clinically as the different forms of icterus gravis and acute yellow atrophy of the liver. Manifestly the term is not strictly applicable to all of these.

Etiology.—The causes of acute congestion of the liver already mentioned may cause acute non-suppurative hepatitis; the difference between these disorders is one of degree only. As a matter of fact, acute hepatitis is uncommon in temperate climates. In the tropics, however, a disorder described by those of experience as acute parenchymatous hepatitis (tropical liver) is of frequent occurrence; it is most commonly provoked by malaria, but also by dysentery and perhaps other infections. It occurs especially in young adult Europeans, and not uncommonly soon (during the first year) after their arrival in the tropics. Undoubtedly factors other than malaria and dysentery are active in the etiology, although these may be the exciting causes acting upon a liver reduced in vitality. The influence of immoderate eating (especially of animal food), of drinking (especially alcohol), and of exposure to cold and fatigue may not be overlooked. These induce an active congestion of the liver, and on account of the consequent increased functional demands reduce its vitality, and perhaps in this way render the liver especially vulnerable to the deleterious influence of the causes of malaria, dysentery, etc.

Pathology.—The lesions are most diverse; they include combinations of congestion, cloudy swelling, focal necroses, and the vascular and other phenomena of inflammation. The lesions vary from small focal necroses to more widespread, if not general, involvement of the liver. In slight or moderate cases the appearances of the liver are those of active

congestion and cloudy swelling; in the more severe cases they vary in grade up to those of acute yellow atrophy. In some (protracted) cases, looked upon by many authors as a subacute form of acute yellow atrophy, there is more or less compensatory hyperplasia of the liver cells.

Symptoms.—The symptoms virtually are those of active congestion of the liver, of which acute hepatitis is only an advanced stage. Perhaps, although not necessarily, the symptoms are more aggravated in the inflammatory than in the merely congestive disorder: the liver pain and tenderness are likely to be more marked; nausea and vomiting are more common and usually more severe; the vomited matter partakes of the so-called bilious character (greenish, watery), and not infrequently contains blood—due usually to violent and distressing retching that provokes marked thirst and ultimately may induce marked prostration or collapse; the bowels are likely to be constipated, but the constipation may alternate with a foul, ill-smelling diarrhœa; and the nervous symptoms, such as headache, restlessness, irritability, mental depression, disturbed sleep, etc., are usually more marked; slight jaundice may develop. The liver becomes enlarged—sometimes notably enlarged. In malarial cases the spleen also becomes enlarged. Fever (101° to 103° F.) supervenes and serves to distinguish the inflammatory from the merely congestive disorder.

Diagnosis.—The diagnosis is usually apparent from the etiological factors and the general symptoms. The diagnostic difficulties pertain to excluding other disorders, such as the different forms of suppurative hepatitis, and in the presence of jaundice, the different forms of toxic jaundice and acute yellow atrophy. In the early stages it is frequently quite impossible to differentiate between suppurative and non-suppurative hepatitis; in some cases time alone will bring the diagnosis. In some cases the one is a later stage of the other, and the suppuration may not be susceptible of recognition until the development of the general phenomena of the condition—irregular chills, fever, sweats, etc. In acute yellow atrophy the constitutional and nervous symptoms are more severe, the liver, at first enlarged, becomes small, the duration is less, and the outcome is usually death.

Prognosis.—The first attack of acute non-suppurative hepatitis, especially as observed in the tropics, usually lasts about a week or ten days, and results in recovery; in some cases, however, suppuration ensues. The one attack, however, predisposes to subsequent attacks, and with each recurrence the course and termination are less favorable.

Treatment.—The treatment of acute non-suppurative hepatitis (as well as of its antecedent stage—active congestion) comprises the avoidance of all factors likely to produce it: therefore, wisdom and moderation in eating and drinking; in the tropics, prophylaxis of the infections known to involve the liver (malaria, dysentery, etc.). When the attack has developed, the patient should go to bed and be kept there until convalescence is complete. The guiding principle should be to reduce the functional demands upon the liver, to eliminate all factors that

might provoke or add to its congestion, and to reduce any existing intestinal catarrh. These objects are best achieved by dietary regulations. The diet should be minimal in amount and absolutely unirritating; in general it should consist exclusively of milk, which should be given at stated (rather long) intervals, and well diluted with lime water, Vichy, or other alkaline water. Whey, junket, albumen-water, strained broths (without seasoning), barley water, calf's foot jelly, etc., may be given later. The bowels should be thoroughly evacuated, preferably by saline cathartics; later, by calomel or blue mass. Ammonium chloride (60 grains, 4 grams, daily) is extensively used by physicians in the tropics, and is believed to control the congestion and prevent inflammation of the liver, to relieve the intestinal catarrh, and to promote the elimination (by diuresis) of retained waste products (urea, uric acid, etc.). It may be given in the early stages, when it should be combined with the alkalies (alkaline waters, sodium bicarbonate, etc.), or later, when it may be combined with nitrohydrochloric acid (highly extolled in hepatic torpidity), nux vomica, or strychnine, and a bitter tonic (gentian, columba, quassia, etc.). The distressing gastric irritability of the early stage of some cases may be controlled by sodium bicarbonate, bismuth subnitrate, and carbolic acid or creosote. Intestinal antiseptics may be useful: β-naphthol, bismuth, resorcin, salol, etc. These subject to exacerbations or recurrences of acute congestion of the liver are much benefited by treatment at such spas as Carlsbad, Marienbad, Vichy, Ems, Saratoga, etc.

Suppurative Hepatitis.—Suppurative hepatitis (abscess of the liver) occurs under a variety of circumstances and in several forms. There is some justification for the common division into two classes—the large, single, so-called tropical abscess, and the small, multiple, non-tropical abscesses; but the processes are not altogether different, since the tropical abscess may be double instead of single, and even when single, by infecting adjacent liver tissue, may give rise to the development of many secondary abscesses, and a number of small abscesses by peripheral extension may eventually coalesce and give rise to a large single abscess.

Etiology.—Abscess of the liver is always the consequence of microorganismal infection; the microörganisms are various, and they reach the liver by one of several pathways. The common causative microörganisms are the ordinary staphylococci and streptococci, Amœba coli (Entamœba dysenteriæ), Bacillus coli communis, Bacillus pyogenes fœtidus, Bacillus typhosus, Bacillus dysenteriæ, Bacillus pyocyaneus, Diplococcus pneumoniæ, Proteus vulgaris, Actinomyces bovis, etc. The infecting agent or agents may reach the liver directly, as when, in consequence of traumatism, bacteria are introduced directly into the liver, or when they reach the liver by extension of disease of adjacent organs; or the infection may be carried from afar by the blood stream, that is, by the portal vein (dysentery, and other causes of suppurative pylephlebitis); by the hepatic artery (general septicopyemic processes); by the hepatic veins (retrograde embolism); by the biliary ducts (suppurative cholangitis and pericholangitis); or by the lymphatics.

Pathology.—The liver is enlarged, swollen, and opaque, and presents the ordinary evidences of parenchymatous degeneration or cloudy swelling. In addition, in the cases of so-called multiple pyemic abscesses, it exhibits a number of variously sized abscesses—usually small, but often coalescing to form larger, sometimes many-chambered cavities, with purulent contents. The so-called single, tropical, or amœbic abscess of the liver is described by Manson as consisting at first of one or more grayish, ill-defined, anemic, circular patches, half to one inch or thereabouts in diameter, in which the lobular structure of the liver cannot be made out. A drop or two of reddish, gummy pus may be expressed from the necrotic patches. Later, the centre of these patches liquefy, and distinct but ragged abscess cavities are formed. An abscess thus commenced extends partly by molecular breaking down; partly by more massive necrosis of portions of its wall; partly by the formation of additional foci of softening in the neighborhood and subsequent breaking down of the intervening septa. These may involve almost if not an entire lobe, usually the right. The pus is usually thick, viscid, chocolate-colored, and streaked with blood, and often contains large pieces of necrotic liver tissue. Entamœba dysenteriæ may be found in more than one-half of the cases (in some of the other cases in the wall of the abscess). The ordinary pyogenic organisms are rarely encountered. A large abscess may consist of a suppurating hydatid cyst.

Symptoms.—*Multiple septicopyemic abscesses*, whether due to portal-vein, hepatic-artery, hepatic-vein, or biliary-duct infection, are often not recognized, since the symptoms of the primary disorder overshadow those due to the infection of the liver. In some cases, however, the spread of the infection to the liver is suggested by aggravation of the general symptoms, especially chills (or chilliness), fever, sweats, and polynuclear leukocytosis, and the development of local symptoms referable to the liver. These consist of pain in the region of the liver, perhaps referred to the right shoulder, tenderness about the liver, a mild grade of jaundice, and enlargement of the liver. The enlargement may be nodular, and the nodules soft and perhaps semifluctuating, and audible and palpable frictions may be detected.

The so-called *tropical abscess of the liver*, although, as a rule, more readily recognized, may be very indefinite in its clinical manifestations, and escape clinical detection. Generally, the onset is insidious: sometimes for weeks or months the patient may complain of general ill health, malaise, or increasing weakness—which are especially significant if they constitute part of the poor health or delayed convalescence following dysentery or other intestinal suppuration or ulceration. In some cases the onset is sudden, attended by a chill. The important symptoms are those of general septicopyemia—chills, fever, sweats, and polynuclear leukocytosis, to which local signs referable to the liver are added. The fever may be quite high (104° to 105° F.), and distinctly intermittent in character. Jaundice occurs in about 16 per cent. of the cases, and the patient may develop a suggestive icteroid facial aspect. The

liver becomes enlarged; in the early stages, when the abscess is still well within the substance of the organ, the enlargement may be tolerably uniform, but as the abscess increases in size, it commonly causes disproportionate enlargement upward, usually of the right lobe—which may assume a characteristic dome shape; that is, the upper limit of hepatic dulness rises near the mammillary line, reaches its highest point (fourth or third rib) near the midaxillary line, and descends, so that in the midscapular line it may be as low as the angle of the scapula. The *x*-rays may disclose the extent and development of the liver enlargement better than percussion. Nausea and vomiting may occur; diarrhœa is rather common, and the patient emaciates. Perforation may occur, especially upward through the diaphragm, the pleura, and the lungs, giving rise to a so-called hepatopulmonary abscess; the expectoration is quite characteristic and has been likened to anchovy sauce. Rupture may occur also into the gastro-intestinal tract, externally, etc.

Diagnosis.—The diagnosis is often difficult; the most suggestive signs consist of one of the several etiological factors, pain in the region of the liver, progressive enlargement and tenderness of the liver, slight jaundice, and chills, fever, sweats, and polynuclear leukocytosis. Leukocytosis, however, may be absent in slow-growing, chronic, and well-encapsulated abscesses. Malaria may be excluded by a history of antecedent dysentery or other etiological factor; evening rise in temperature (that of malaria being more common during the day); disproportionate enlargement of the liver as compared with the spleen (the reverse being the case in malaria); absence of malarial parasites in the blood; the occurrence of polynuclear leukocytosis; and the non-response to quinine. Cholelithiasis and Charcot's biliary intermittent fever may be excluded by the absence of a history of cholecystitis, cholelithiasis, and biliary infection; the absence of attacks of fever, etc., separated by longer or shorter periods of apyrexia; the non-occurrence of increase in the jaundice after each attack; and the absence of serious impairment of the general health despite the long duration of the disorder.

Prognosis.—The septicopyemia, of which the multiple liver abscesses are merely a part, is almost invariably fatal. Many of the so-called single or tropical abscesses result in recovery after operation.

Treatment.—The treatment of the multiple septicopyemia abscesses is that of septicopyemia in general, and is quite as ineffectual. Perhaps treatment by specific bacterins may yet prove of service. The treatment of the large, single abscess is surgical and consists of thorough evacuation and drainage.

CHRONIC INTERSTITIAL HEPATITIS: THE CIRRHOSES OF THE LIVER.

Eliminating from consideration cases of (1) so-called capsular cirrhosis (more properly called perihepatitis or hepatic capsulitis), in which the liver tissue may be not at all affected, except perhaps secondarily in

consequence of compression or by slight ingrowth of new connective tissue from the capsule; (2) so-called cardiac cirrhosis or cardiac liver, which develops in consequence of long-standing passive congestion; (3) syphilis of the liver, in which the lesions are specific and peculiar to that infection; and (4) circumscribed or focal fibroses, which follow limited inflammatory and degenerative processes, focal necroses, chronic obstruction of the biliary ducts (pericholangitis), etc.—there are two types of chronic diffuse disorder of the liver attended by fibrosis, to which the term cirrhosis may be limited. One of these disorders is very common; it is usually due to the misuse of alcohol, or perhaps other poison carried by the portal circulation; it is characterized by moderate enlargement of the liver (which in advanced stages may become reduced in size), by newgrowth of connective tissue, especially conspicuous in and about the portal spaces, by phenomena of portal obstruction (notably hematemesis and ascites), and by the absence or unimportance of jaundice; and after the development of symptoms it usually runs a comparatively short course (Laënnec's, portal, alcoholic, atrophic, or multilobular cirrhosis). The second of these disorders is rare; it is of unknown etiology; it is characterized by marked and persistent enlargement of the liver and spleen, by chronic jaundice, by periodic attacks of abdominal pain and fever, and by the absence of manifestations of portal obstruction (notably ascites); the essential lesion is a radicular cholangitis, and it runs a comparatively long course (Hanot's, biliary, hypertrophic, or monolobular cirrhosis.

In conditions of long-standing obstruction to the large biliary ducts, especially from gallstones, more or less fibrosis develops in and about the smaller biliary ducts in the liver, and may spread to the adjacent liver tissue; the ensuing induration is sometimes spoken of as obstructive biliary cirrhosis (more properly cholangitis and pericholangitis).

PORTAL CIRRHOSIS.

Portal cirrhosis is a chronic degenerative and inflammatory disease of the liver characterized by recurring degeneration and regeneration of the hepatic parenchyma and by concomitant and consecutive fibrosis in and about the interlobular or portal spaces, all of which leads ultimately to obstruction of the portal circulation.

Etiology.—Portal cirrhosis is most common during the fifth decade; although it is by no means rare at the extremes of life. A number of cases occur in young children, in whom it is often fatal before the sixth year. It is a little more than twice as common in men as in women. It is undoubtedly the expression of the activity of some poison or poisons; these are various in nature, and may reach the liver by way of the portal vein, the hepatic artery, or both. In the great majority of cases it follows addiction to alcohol, especially distilled liquors (whiskey, gin, brandy, etc.), which are a direct poison to the hepatic parenchyma, and set up

also gastro-intestinal catarrh; this doubtless results in the formation of divers autotoxins that may injuriously affect the liver. A special form of cirrhosis, believed to be due to highly seasoned food, fermentative dyspepsia, etc., is recognized (Budd's non-alcoholic or dyspeptic cirrhosis). Infections of various kinds, such as malaria, tuberculosis, syphilis, uncinariasis, distomatosis (bilharziosis), etc., may be followed by cirrhosis, but the relationship of the one to the other has not been determined; in most cases the necroses and fibroses are focal, and the lesions are not really cirrhotic in an acceptable sense. Cirrhosis is also associated with a type of hemolysis (hemochromatosis).

Pathology.—The liver varies much in size in different cases, and in the same case at different times. At the necropsy it may be very small, weighing only 980 grams or less; or it may be very large, weighing as much as 4000 grams or more; but it is worthy of note that in the majority of cases at the necropsy it weighs more than normally. It is of increased density, increased consistency, and lessened elasticity. The capsule is often thickened and united to the diaphragm by adhesions. The surface of the organ is distinctly granular, the small livers more so than the larger ones: grayish-white depressions alternate with pale, yellowish-brown, sometimes reddish-brown, roundish or ovoid elevations —the so-called coarsely granular or "hobnail" liver. The cut surface of the liver reveals interlacing bands of whitish or grayish-white fibrous connective tissue that vary somewhat in thickness, pervade the entire organ, and are continuous with the grayish opaque depressions on the surface of the liver. These bands of fibrous connective tissue, which obviously take their origin in the periportal spaces, form a network that encloses or surrounds islets of liver tissue that vary in size from that of a pinpoint to others 1 cm. or more in diameter; the larger islets are made up of several, often eight, ten, or more liver lobules (or remains of liver lobules), and are pervaded by more delicate bands of connective tissue (so-called multilobular cirrhosis). The liver tissue is usually pale yellowish in color or yellowish brown (κιρρός, yellowish); occasionally it is greenish (from staining with bile), or reddish or brownish, especially in the centres of the lobules (from staining with blood or blood pigment). Microscopically, the essential lesions consist of a variable degree of destroyed, atrophic, hyperplastic, and rearranged hepatic parenchyma and of newgrown fibrous connective tissue. In consequence of the activity of some poison, larger or smaller areas of liver cells are destroyed. The surviving cells attempt by hyperplasia to compensate for the cells lost.

The continuing activity of the etiological factor leads to a continuous or recrudescing death of certain old as well as newly formed liver cells, which in turn is followed by renewed regeneration. This continuous degeneration and regeneration ultimately lead to complete transformation of the architecture of the liver, certain lobules are entirely destroyed, others are partly or completely rearranged, some are markedly hyperplastic, although tolerably well arranged, some show dendritic and other irregular proliferation (including the newly formed, so-called, pseudo-bile

ducts), new islets of liver tissue entirely devoid of central veins appear, and, what is especially important, new circulatory conditions—capillary and venous—arise. In some cases there is considerable fatty infiltration of the liver cells (so-called fatty cirrhosis).

Concurrent with the degeneration of the liver cells, the interstitial connective tissue early shows more or less round-cell infiltration and other inflammatory phenomena, especially in and about the portal spaces; later there is considerable increase in the amount of connective tissue—in part the result of organization of the inflammatory exudate, in part a replacement fibrosis to fill up potential lacunæ left by destroyed liver parenchyma, in part the tissue originally between the lobules (the connective tissue of Glisson's capsule), and in part the collapsed and otherwise altered reticulum of certain liver lobules the cells of which have disappeared. This newly formed connective tissue commonly encloses rather than invades the liver lobules; sometimes only one lobule is enclosed in a wide band of connective tissue, sometimes many lobules—whence the designation multilobular cirrhosis. There is usually also considerable increase in the elastic tissue. This fibrosis ultimately contracts, and on the one hand causes atrophic changes in the liver cells, and on the other, obstruction and obliteration of some of the interlobular and intralobular branches of the portal vein.

The spleen becomes enlarged in 80 per cent. of the cases—the consequence of toxic hyperplasia and of passive congestion. The gastrointestinal tract becomes congested and chronically inflamed, and reveals many dilated and tortuous veins, which, rupturing, give rise to more or less severe hemorrhage. The kidneys may reveal chronic interstitial changes. The heart and the pancreas often become fibrosed. The peritoneum is commonly thickened, and not infrequently becomes the seat of miliary tuberculosis (which may determine the final lethal issue).

Communications between the portal vein and the systemic circulation constitute a part of the cirrhotic process. For the most part these consist of dilatations of normally existing anastomoses. The important are (Rolleston): (1) A general anastomosis between the veins of the peritoneum and those of the abdominal wall, such as the lumbar and renal. (2) Those around or in connection with the liver, especially the paraumbilical veins (Sappey) of the falciform ligament, which put the portal vein in communication with the veins of the anterior abdominal wall. This anastomosis may show itself as a "caput medusæ," or a number of dilated veins around the umbilicus. This must be distinguished from the more marked venous anastomoses which result from obstruction to the passage of blood along the inferior vena cava; in the latter the dilated epigastric and mammary veins avoid and do not centre around the umbilicus. (3) Between the œsophageal veins, discharging into the azygos veins and thence into the superior vena cava, on the one hand, and the gastric veins, on the other. These veins may become varicose (œsophageal varices or piles), and are a common cause of profuse and

even fatal hemorrhage. (4) Between the superior hemorrhoidal veins, tributaries of the inferior mesenteric vein, and the middle and inferior hemorrhoidal veins which open into the internal iliac veins (the cause of piles). These anastomoses serve both a useful and an evil purpose. On the one hand, as long as the collateral circulation is efficient, obstructive symptoms are in abeyance and the disordered liver is enabled better to perform its functions, perhaps enabled to regenerate, whence in some cases the disease remains or becomes latent, even apparently cured; on the other hand, (*a*) the dilated veins are likely to rupture, either spontaneously or in consequence of trauma, and lead to more or less severe hemorrhage (hematemesis, enterorrhagia, hematoma of the abdominal wall about the round ligament); and (*b*) much of the blood from the gastro-intestinal tract being thus diverted from the liver to the general circulation, conditions somewhat analogous to those induced by the establishment of an Eck's fistula (the experimental communication between the inferior vena cava and the portal vein), supervene, much of the products of digestion is not brought under the influence of the liver, is not detoxified, and toxemia ensues.

Symptoms.—Cirrhosis of the liver may remain for many years entirely latent; indeed, in most cases the lesions in the liver are well advanced before the onset of noteworthy symptoms, and the disease not infrequently constitutes an unexpected finding at the necropsy table. Symptomatically, the disease has been divided into the preascitic and the ascitic stage, and the symptoms are described as being (1) obstructive, and (2) toxic. As long as the collateral circulation is efficient, obstructive symptoms are in abeyance.

In a few cases, during the early stages of the development of the disease, phenomena attributed to an ill-defined, so-called, active hyperemia of the liver are encountered; that is, the patient complains of ill-defined or vague gastric symptoms, a sense of weight in the right hypochondrium, perhaps short transitory attacks of slight fever, slight jaundice, and actual pain in the region of the liver; and the liver on examination is ound to be enlarged and tender. Such attacks may last several days, disappear, and recur at longer or shorter intervals.

In most cases, however, the early symptoms are an expression of gastro-intestinal catarrh—due partly to portal congestion, and partly, in many cases at least, to chronic alcoholism with acute exacerbations. The patient complains of coated tongue, irregular appetite, flatulence, epigastric distress, especially after eating, sometimes of nausea and vomiting (often in the morning, and usually referable to alcoholism), irregular action of the bowels (alternating attacks of diarrhœa and constipation), or habitual constipation, hemorrhoids, etc. The increasing portal obstruction leads to increasing congestion of the gastro-intestinal tract and the spleen, the congestion being doubtless favored by the absence of valves in the portal tributaries. This congestion reduces the functional capacity of the stomach and intestines, limiting the digestive juices and the intestinal secretions (although sometimes increasing the mucus),

reducing the absorbing power of the intestines, and often inhibiting peristalsis. Constipation increases and tympanites not infrequently ensues, and both may become of high grade and distressing; occasional relief is sometimes afforded by attacks of diarrhœa which relieve the engorged vessels. These phenomena, while not diagnostic, are highly suggestive of the disease, especially in association with a history of alcoholism.

With the progress of the disease the foregoing evidences of derangement of the gastro-intestinal tract become more marked, and the general health becomes impaired; but the first obtrusive manifestation of the disease, that which induces the patient to seek medical advice, is not infrequently hematemesis or ascites.

Hematemesis is an initial symptom in some cases, and it is a common event some time in the course of most cases. Usually it occurs early—while the liver is large; when not the initial symptom, it is often the first evidence of serious disease (33 per cent. of the cases); but it may occur late in the disease, that is, after the development of ascites and when the liver may have become small. With the hematemesis there is usually blood in the stools; but blood may occur in the stools without the vomiting of blood, especially when the bleeding into the stomach is slow, and also when, as is not uncommon, the source of the blood is the intestine or the rectum or hemorrhoids. In suspected cases of cirrhosis examination of the stools for blood (even occult blood) should be early resorted to. Hemorrhage from or into other parts of the body is very common. especially epistaxis, bleeding from the gums, larynx, lungs, and into the skin and mucous membranes.

Jaundice is not a part of portal cirrhosis, but it occurs as a complication some time during the course of from 15 to 20 per cent. of the cases, and is usually due to radicular cholangitis.

Toxic symptoms may develop at any time during the course of the disease. Minor symptoms consist of restlessness, weakness, headache, and itching; the itching may precede or be unassociated with jaundice. More severe toxic symptoms comprise: (1) Apathy, stupor, and coma; (2) an active noisy delirium; and (3) convulsions, paralyses, contractures, one of which may alternate with the others.

Cirrhosis of the liver may be afebrile throughout its course; during the terminal stage the temperature may be subnormal; but attacks of fever (100° to 102.5° F.) are not uncommon. Sometimes these seem to be part of the disease, especially when it advances rapidly; sometimes they are due to attacks of perihepatitis; sometimes they are due to complications. The more continuous fever, which is rather common late in the disease, is usually due to tuberculosis—of the peritoneum, lungs, etc.

Physical Signs.—The abdomen is more or less markedly, sometimes enormously, distended, and is in notable contrast to the emaciated, subicteroid face and extremities. The abdominal wall reveals enlarged and varicose veins due to the collateral circulation. The liver is enlarged in most cases, but the size varies considerably in different cases and in the

same case at different times. Its surface may be distinctly rough to palpation ("hobnails"). In some cases late in the course of the disease the liver becomes considerably reduced in size. The spleen is usually enlarged, palpable two or three fingers' breadth below the left costal margin. It is often much reduced in size by copious gastro-intestinal hemorrhage, severe diarrhœa, the recurrence of ascites after tapping, etc.

Ascites is often the first obtrusive manifestation that leads the patient to seek medical advice. It is usually a late event. In most cases the liver is larger than normally at the time of development of the ascites; but a large liver may be present without ascites; a small liver is rarely found without ascites. The fluid varies in amount—in the average case from 4 to 8 or 10 liters, but it may be as much as 20 liters. It is due to different causes: (1) Portal obstruction, the result of lessening of the portal area in the liver, and the communication of the hepatic-artery pressure to the portal-vein radicles; (2) a complicating perihepatitis or more widespread peritonitis, which is very common in the late stages of the disease; and (3) toxemia; the toxic agent is not known, but it is believed to act as a lymphagogue on the peritoneum. Varying with the amount of fluid, there is more or less displacement and compression of the abdominal and thoracic organs. The liver and the diaphragm are pushed upward and cause embarrassment of the respiration and the heart action, and not infrequently compression and consequent congestion and collapse, and perhaps bronchopneumonia, of the bases of lungs, particularly the right. Œdema is common late in the disease, occasionally it may precede the ascites, in which event it is toxic, anemic, cardiac, or renal in origin. The blood shows the changes of secondary anemia. There is no leukocytosis unless there is some associated infection, which is not rare. Late in the disease evidences of considerable toxic disorganization of the blood become apparent—hydremia, œdema, submucous and subcutaneous hemorrhages, etc., and the blood findings rarely may simulate those of pernicious anemia.

Complications.—Infections of various kinds are common, especially tuberculosis—of the lungs, pleura, or peritoneum. Tuberculous peritonitis is very common, and not infrequently determines the fatal issue. It is suggested by fever, tenderness and rigidity of the abdominal wall, perhaps increase in the amount of ascites previously stationary (the fluid may become hemorrhagic), more rapid course, and speedy death. Chronic nephritis occurs in about one-half of the cases.

Diagnosis.—The diagnosis of cirrhosis of the liver in the early stages is not always an easy matter, but it should be made more frequently than it is. In an alcoholic subject with dyspepsia, an enlarged and tender liver suggests the diagnosis; and this is virtually confirmed if to these there be added an enlarged spleen, perhaps recurring attacks of pain in the region of the liver, with slight fever or slight jaundice, and urobilinuria. Later, when hematemesis, ascites, the hepatic facies, etc., have developed, the diagnosis can be no longer in doubt. When the liver is enlarged and there is no ascites, one must exclude other causes of enlargement of the liver,

such as passive congestion, fatty liver, amyloid liver, leukemia, malaria, and syphilis. The occurrence of hematemesis and other gastro-intestinal hemorrhages necessitates differentiation from gastric and duodenal ulcer. When ascites has developed, one must exclude other causes of ascites, such as chronic peritonitis and perihepatitis (multiple serositis), thrombosis of the portal vein, tumors of the peritoneum and abdominal organs, chronic cardiac disease, and cachectic states. Splenic anemia (Banti's disease) also must be considered in some cases.

There are several special types of portal cirrhosis, of which the following are the most important: (1) Hemochromatosis (bronzed diabetes), a disorder that occurs especially in male adults, and is characterized by pigmentation of the viscera and usually also of the skin; cirrhosis of the liver; and fibrosis of the pancreas, with involvement of the islands of Langerhans and consequent diabetes. The pigmentation is usually attributed to increased hemolysis. The noteworthy symptoms consist of general cutaneous pigmentation (suggesting Addison's disease), enlargement of the liver and spleen, and the ultimate development of diabetes. Ascites may be slight or absent; hemorrhages may or may not occur, but death may result from rupture of œsophageal varices. Diabetes, when it supervenes, is severe and dominates the clinical phenomena; acidosis is common and usually leads to coma. (2) Malarial cirrhosis is said by those of experience to occur in the tropics. (3) Anthracotic cirrhosis is very rare. (4) Cirrhosis in children is usually of the portal type. The symptoms in general conform to those in the adult. Hematemesis is less common (greater distensibility of the spleen in children), diarrhœa and slight jaundice are more common; the liver and the spleen are disproportionately larger than in adults (relatively greater volume and more ability for repair in children); tuberculous peritonitis is common, and perhaps accounts for most of the ascites and the not uncommon fever.

Prognosis.—The prognosis is not encouraging. Ordinarily the disease runs a fatal course within three years from the onset of symptoms. Doubtless if the disease were earlier recognized, arrest could be secured in many cases.

Treatment.—The only trustworthy treatment is based upon our conception of the etiological factors, and consists essentially in the avoidance of all causes known or suspected to lead to the disease. Of these, the most important are alcohol, stimulating and highly seasoned foods, foods likely to undergo fermentation in the intestinal tract, etc., all of which must be strictly prohibited. Milk is the best diet for a month or six weeks, and from time to time alkaline waters may be added to it. Later, one may give also eggs, gruels, cereals, stewed fruits, fish, and green vegetables, and some meat from time to time. Good results sometimes follow the prolonged use of the iodides, to which minute doses of mercuric bichloride may be added. Nitrohydrochloric acid and ammonium chloride are said to be efficacious in some cases. Indigestion, nausea, vomiting, hematemesis, constipation, diarrhœa, etc., are to be treated

as mentioned under those headings The ascites, if not relieved by purgatives (blue mass or calomel and saline cathartics), diuretics (alkaline diuretics, theobromine, spartein, caffeine, apocynum cannabinum), the Niemeyer pill (page 477), and a dry diet, should be tapped. The Talma-Morrison operation (omentopexy, epiplopexy) is of service in carefully selected cases early. It is designed to relieve the portal circulation, by diverting the portal blood to the general systemic circulation through the medium of adhesions set up between the liver, the omentum, and the parietal peritoneum. Associated cardiac, pulmonary, or renal disease, repeated tappings, jaundice, toxemia, and emaciation are contra-indications to performing operation.

BILIARY CIRRHOSIS.

Biliary cirrhosis is a chronic disorder of the liver, probably infectious in origin, characterized anatomically by radicular cholangitis and connective-tissue hyperplasia, and clinically by chronic jaundice and enlargement of the liver and spleen.

Etiology.—Biliary cirrhosis is especially common during adolescence and early adult life; most cases occur between the twentieth and the thirtieth year; it is rare after the fortieth year, but it is quite common in children (juvenile type), especially in India. In adults about 85 per cent. of the cases occur in males; in children it is equally common in the two sexes. A seeming hereditary influence is not uncommon.

The exact nature of the disease is not known. Some, but not all, subjects are alcoholic; and, on the whole, it seems that alcohol plays at most a minor etiological role. In the cases so common in children in India it is not in any way an etiological factor. In other cases, perhaps, it reduces the resisting power of the liver and thus permits of the more ready operation of other causes. There is some inconclusive evidence that the disorder is the consequence of a general blood infection that provokes a radicular cholangitis; but no definite etiological agent has been discovered. An infection is suggested by the disorder sometimes developing after infectious diseases, by the common occurrence of attacks of fever and leukocytosis, and the not rare occurrence of general lymph-node enlargement.

Pathology.—The liver is enlarged and weighs from 2000 to 4000 grams; its shape is maintained, its consistency increased, and its elasticity diminished. The surface is smooth and exhibits very fine granulations (in marked contrast to the coarse granulations of portal cirrhosis). The liver is usually dark olive green in color. The cut surface is dark greenish or yellowish green in color, smooth, or very slightly granular in advanced cases; usually the liver lobules are more or less obvious, sometimes apparently enlarged, and always separated by widened trabeculæ of connective tissue. Microscopically, the obtrusive feature consists of an overgrowth of fibrous connective tissue, which occurs in delicate fibrillar

bands that not only course between the lobules, but also invade the lobules to a greater or less extent (so-called monolobular, unilobular, or intralobular cirrhosis). In some advanced cases this arrangement of connective tissue is not so regular. The basic and essential lesion of the disorder, however, seems to be a radicular cholangitis—which is disclosed by proliferation and desquamation of the epithelium of the small biliary ducts. This leads to obstruction and consequent dilatation of the biliary canaliculi, which in turn are found distended with inspissated bile and bile thrombi. Branching columns of cells, so-called pseudobiliary canaliculi, in and about the periphery of the liver lobules (due to hyperplasia of the liver cells as well as of preëxisting ducts), constitute a noteworthy feature of the lesions. The liver cells exhibit rather inconspicuous lesions, but acute necrotic autolytic changes may occur at any time and determine the fatal issue. All the organs of the body are bile-stained. The spleen is enlarged, and commonly weighs 600 to 1500 grams or more; in children it may be as large as, if not larger than, the liver. The lymph nodes in the portal fissure are enlarged, œdematous, and congested, but they exert no pressure on the biliary ducts. Occasionally the lymph nodes in other parts of the body are similarly, although less, affected.

Symptoms.—The onset of the disease is insidious; for a long time there may be ill-defined complaint of poor health, general malaise, weakness, loss of flesh, etc.; or there may be a sense of weight or distress, perhaps actual pain, in the right hypochondrium; rarely there is some dyspepsia, coated tongue, poor appetite, perhaps nausea, diarrhœa, etc.; in a few cases the patient's attention may be first directed to increase in the size of the abdomen.

In the great majority of cases the first obtrusive symptom is jaundice. This varies in degree from time to time, periods of exacerbation and of regression alternating; but, on the whole, it tends to increase with the progress of the disease, and eventually it may become extreme, usually greenish. The usual concomitants of protracted jaundice are seen; there is often intense itching, eczema, lichen, sometimes xanthelasma, etc.; bleeding from the nose or the gums or other mucous membranes may occur from time to time for a long period, or there may be recurring attacks of purpura; the urine shows the presence of bile pigment. The feces also contain bile pigments and may be very dark (in contrast to the clay-colored stools of obstructive jaundice).

In addition, the disease is characterized by periodic attacks of more or less severe abdominal pain, especially in the region of the liver, sometimes attended by nausea and vomiting, and associated with fever, leukocytosis, and increase of the jaundice. These attacks occur without regularity and without demonstrable cause, and they last variable periods —several days to several weeks. Usually the fever does not go above 102°; in some cases there is slight fever for long periods. Occasionally these exacerbations are attended by the nervous and other phenomena of icterus gravis, and with high fever, delirium, coma, etc., the patient may die.

Examination reveals the liver to be uniformly enlarged, reaching sometimes to or below the transumbilical line; it presents a smooth or finely granular surface. The spleen also is enlarged, much more so than in portal cirrhosis. It is said that the enlargement of the spleen sometimes antedates that of the liver. In children the spleen may be actually larger and heavier than the liver—the juvenile type of biliary cirrhosis (cirrhose biliaire splénomégalique of Gilbert and Fournier). The blood reveals oligocythemia, leukocytosis (increased at the time of the periodic exacerbations), and diminished coagulability.

Rarely, a bulbous enlargement involving the soft tissues of the fingers and toes (Hippocratic fingers) occurs. In children, growth is stunted, a type of infantilism develops, and the onset of puberty is delayed.

Diagnosis.—The important diagnostic features consist of the occurrence in a young, usually non-alcoholic subject of chronic progressive jaundice, with considerable and persistent enlargement of the liver and spleen, and periodic attacks of abdominal pain, fever, and leukocytosis, with subsequent increase in the jaundice, and no sign of cholelithiasis or portal obstruction. There should be no difficulty in differentiating the more common or portal cirrhosis—which occurs, as a rule, in older and alcoholic subjects and is attended by more marked dyspeptic symptoms and by evidences of portal obstruction (dilatation of the subcutaneous veins of the abdomen and ascites), which are absent in biliary cirrhosis. In portal cirrhosis, hemorrhages, especially hematemesis, often occur early and are commonly profuse; whereas in biliary cirrhosis hemorrhages are usually a late manifestation and small. Jaundice sometimes occurs in portal cirrhosis, but it is a complication and catarrhal in nature; in biliary cirrhosis it is the conspicuous feature, and, although permanent, it is not associated with absence of bile from the feces. Portal cirrhosis usually runs a fatal course within three years or less; biliary cirrhosis not uncommonly lasts from five to ten years or more.

Prognosis.—The outlook as regards cure is hopeless, but the disease may last for many years and the general health may be well maintained.

Treatment.—The treatment is largely symptomatic. The hygienic conditions should be the best obtainable. The diet should be generous, but dietetic indiscretions, alcohol, etc., should be avoided. The probable infectious nature of the disorder has suggested the use of antiseptics, such as the salicylates and hexamethylenamin, which are eliminated with the bile; apparent improvement has been reported in some cases. Good results are said also to have followed drainage of the gall-bladder and the biliary ducts, but the diagnoses in some of these cases is open to question.

TUMORS OF THE LIVER.

Etiology.—Carcinoma of the liver may be primary or secondary (to carcinoma of the stomach, pancreas, intestine, rectum, female genitalia, breast, etc.). A primary growth is very rare, the secondary being

from twenty-five to forty times as common. Both occur in adult life and old age. The primary tumor seems to be slightly more common in men than in women, the secondary slightly more common in women. Sarcoma and so-called benign growths are rare.

Pathology.—Primary carcinoma appears under three forms: (1) Nodular or multiple carcinoma (65 per cent. of the cases)—numerous grayish-white nodules scattered throughout the liver; (2) massive carcinoma (23 per cent. of the cases)—a large mass occupying almost, if not quite, an entire lobe; and (3) infiltrating or diffuse carcinoma (12 per cent. of the cases)—so-called cirrhosis with multiple adenoma or carcinoma. The growth may develop from the hepatic cells or the biliary epithelium. In some cases the sequence of events is represented by hepatic-cell hyperplasia, adenoma, carcinoma. Secondary carcinoma appears as multiple grayish nodules scattered throughout the organ; these often become necrotic, softened, and umbilicated. Primary sarcoma may occur as a nodular or a diffuse growth; secondary sarcoma is usually melanotic in type, and follows a primary growth in the choroid coat of the eye.

Symptoms.—The symptoms do not differ notably whether the growth be primary or secondary, and carcinomatous or sarcomatous. Usually the onset is insidious; the patient may complain of general ill health, languor, anemia, emaciation, etc., for a long time before the true nature of the disorder is disclosed; in many cases, certainly in at least one-third of the cases of secondary carcinoma, the actual conditions are recognized only at autopsy. In some cases, symptoms referable to the primary growth precede, for a variable time, symptoms significant of involvement of the liver; but in a considerable number of cases, carcinoma of the stomach, rectum, etc., may be for a long time latent, or the symptoms are not obtrusive, and the first noteworthy manifestations may be referable to the liver. In a small percentage of cases the primary growth remains insignificant; the lesions are most marked in the liver, and the symptoms almost, if not exclusively, hepatic. They consist of local pain and distress, jaundice, ascites, and enlargement of the liver. The local discomfort (or sense of weight) is usually proportionate to the size and rapidity of growth of the carcinoma; in some large carcinomas, however, there may be no actual pain. Jaundice occurs in more than one-half of the cases; it varies in degree at first, but tends to increase and to become permanent. If a large duct becomes obstructed, the stools become more or less pale, perhaps even clay-colored. Progressive enlargement of the liver is the phenomenon that determines the diagnosis. The enlargement may be moderate or great; it usually involves especially the right lobe, and it is characteristically nodular—the nodules often becoming umbilicated (softening, necrosis, or hemorrhage). Ascites occurs in about 20 per cent. of the cases, being due to portal obstruction or perihepatitis.

In the course of time secondary nodules may develop along the falciform ligament and become palpable through the linea alba or at the umbilicus, or in consequence of penetration of a branch of the hepatic vein, more distant metastases may supervene (lungs, etc.); the patient

loses strength, and becomes anemic, emaciated, and cachectic; and phenomena attributable to hepatic insufficiency (cholemia) may develop.

Diagnosis.—The diagnosis is not always possible, especially in the cases of secondary growths in which the deposits are few in number, small in size, and deep within the substance of the liver. In cases in which a primary growth is obvious or found on examination, and in which there is persisting severe jaundice, emaciation, cachexia, and a rapidly enlarging and nodular liver, the diagnosis can often be made on inspection alone, especially if the patient be made to take a deep inspiration (whereupon the irregular nodular liver, covered only by a thin layer of skin, will be seen to descend). In the differential diagnosis one must exclude tumors of adjacent organs, as well as other causes of enlargement of the liver, especially cirrhosis, syphilis, abscess, and hydatid disease.

Prognosis.—The outlook is hopeless. The average duration of life after the development of symptoms referable to the liver is seven months.

Treatment.—The treatment is purely palliative and symptomatic.

DISEASES OF THE GALL-BLADDER AND BILIARY DUCTS.

CONGENITAL OBLITERATION OF THE BILIARY DUCTS.

Congenital obliteration of the biliary ducts is a disorder characterized anatomically by obliteration or atresia of the biliary ducts and biliary cirrhosis of the liver, and clinically by persistent jaundice and so-called cholemia, which proceed to a fatal issue. The nature of the disorder has not been definitely determined, at least for all cases. It may be the consequence of an anomaly of development, or it may be due to syphilis, to fœtal peritonitis and adhesions, or to a form of hepatic cirrhosis with descending and obliterative cholangitis. Clinically, the disorder is characterized by jaundice, which is present at or develops soon after birth, although rarely it may be delayed for several months; it becomes persistent and intense. The stools become acholic, if not acholic from birth; the urine contains bile pigments; the liver and the spleen are enlarged; vomiting often ensues; and hemorrhages occur from the umbilical cord and into the subcutaneous and the submucous tissues. Gradually the infant emaciates; stupor, coma, or convulsions (cholemia) develop, and death ensues. The course of the disorder is rarely less than a week; usually it lasts for several weeks or several months. The more benign icterus neonatorum may be distinguished usually by the presence of bile in the feces and by the less marked jaundice which soon fades. Syphilis may be excluded by the absence of a history of syphilis in a parent, the absence of other evidences of syphilis, and the non-response to antisyphilitic treatment.

ACUTE CATARRHAL CHOLANGITIS.

(*Catarrhal Jaundice.*)

Etiology.—Acute catarrhal cholangitis is a common disorder in young, especially male, adults, although it occurs at all ages. It is usually due to dietetic indiscretions, such as eating too much food, or indigestible or tainted food, or overindulgence in alcohol—which set up a duodenal catarrh, which spreads to the biliary papilla and causes swelling of the mucosa and obstruction to the free flow of bile. Similar conditions sometimes occur in infections like pneumonia, typhoid fever, etc., in passive congestion of the duodenum, etc. Epidemics are sometimes observed.

Pathology.—The lesions consist of congestion, swelling, œdema, and increased production of mucus at the lower end of the common bile duct and of the duodenal mucosa adjacent to the diverticulum of Vater. In some cases the swelling itself is sufficient to obstruct the flow of bile, but in other cases a plug of inspissated tenacious mucus effectually blocks the diverticulum of Vater. Eppinger found hyperplasia of the lymphoid tissue of the mucosa of that part of the common biliary duct that runs in the wall of the intestine; this had led to complete occlusion of the common duct and dilatation of the rest of the biliary system.

Symptoms.—In most cases the early symptoms are those of the provoking gastro-intestinal catarrh; loss of appetite, bad taste in the mouth, coated tongue, foul breath, epigastric distress, especially after eating, flatulence, nausea, and perhaps vomiting; there may be constipation, or perhaps diarrhœa from extension of the intestinal catarrh; and the patient complains of headache, vertigo, mental depression, general malaise, etc. Rarely there is an acute onset, jaundice being the first symptom noticed; but in most cases after the mentioned symptoms have lasted for a few days to a week, the patient notices, or his attention is directed to the fact, that he is slightly jaundiced. The jaundice is of slow and insidious onset, and gradually increases. With the appearance of the jaundice, sometimes before there is any obvious jaundice, the stools become pale in color and soon are devoid of bile pigment; the urine becomes scanty, dark in color, deposits an abundant sediment, and shows the presence of bile pigments. Usually the bile pigments can be detected in the urine before the appearance of the jaundice in the sclerotics. Pruritus is often marked; sometimes it even precedes the development of the jaundice. The itching is sometimes so extreme as to induce severe eczema, in consequence of uncontrollable scratching. The pulse usually is slowed, 60 or less per minute, and may reveal the dicrotism of low vascular tension.

In most cases neither the liver nor the gall-bladder is palpably enlarged or tender. Fever (101°) is occasionally present, but it is rarely due directly to the cholangitis, except in the epidemic infectious cases; that which sometimes occurs at the beginning of the attack and lasts a day or two is probably due to the gastro-enteritis; when conspicuous or when

it lasts more than several days, it suggests some one of the types of toxic or infectious jaundice (of which, in reality, it may be a mild manifestation).

At the end of about a week, sometimes not until later, the evidences of gastro-intestinal irritation subside; gradually the evidences of biliary obstruction lessen, bile pigments appear in the feces, and disappear, first from the urine and then from the skin, where, however, traces may be found for several months. Restoration of the general health is often much delayed; although the appetite may return at the end of a week or two, improvement in the general health does not occur until the bile pigments begin to be removed from the tissues.

Diagnosis.—In most cases the diagnosis is readily made from the youth of the patient, an antecedent gastro-intestinal catarrh provoked by some more or less obvious cause, and the absence of local pain and of serious involvement of the general health (emaciation, etc.); the diagnosis is further confirmed by the benign and favorable course of the disorder, which in many cases definitely determines the diagnosis. Doubt regarding its catarrhal nature should always be awakened by the occurrence of jaundice in a middle-aged or elderly person, if it becomes deep or olive-tinted, and if it lasts more than six weeks; this is much more likely to be due to impaction of a gallstone or to carcinoma compressing the common duct.

Prognosis.—As a rule, the prognosis is good; the disease of itself does not endanger life, and recovery is usually complete and lasting at the end of three to four or six weeks.

Treatment.—The treatment is essentially that of the gastro-intestinal catarrh, commonly provocative of the cholangitis. The diet should be reduced and for several days should consist of a moderate amount of milk, preferably skimmed milk, or broth. The natural alkaline waters are serviceable; but alkalies, especially sodium bicarbonate, may be added to ordinary water. The bowels should be opened freely. Custom sanctions the use of calomel, which may be given in a single large dose or in repeated small doses (a blue pill is equally efficacious); it should be followed by a saline aperient. They should be given in sufficient dose to cause one or two free evacuations daily; more are undesirable. Injections into the rectum of cold water are sometimes very efficacious in stimulating intestinal, and perhaps also gall-bladder, peristalsis. As the biliary stasis begins to subside, dilute nitrohydrochloric acid and bitter tonics render excellent service. Excessive flatulence is sometimes distressing; it may be relieved by the use of bismuth salicylate, soda-mint, aromatic spirit of ammonia, chloroform water, and antifermentatives; magnesia or rhubarb with sodium bicarbonate and charcoal also are of much service, and relieve the constipation. The pruritus may be relieved by cold sponging, or a carbolic-acid lotion (1 to 40), or the internal use of the bromides.

SUPPURATIVE CHOLANGITIS.

Etiology.—Suppurative cholangitis results from virulent bacterial infection of the biliary ducts. The common infectious agents are Bacillus coli *communis*, staphylococci, streptococci, pneumococci, Bacillus typhosus, Bacillus aërogenes capsulatus, etc. The infection may occur by way of the portal or the general systemic circulation (descending infection), or by way of the duodenum and the diverticulum of Vater (ascending infection). In the great majority of cases there is some local disease of the biliary tract, which acts as an almost necessary predisposing factor; this comprises all disorders that interfere with the free circulation of the bile (that is, cause stagnation), and reduce the resistance of the biliary ducts, especially gallstones and tumors obstructing the duct. Usually there is associated disease of the gall-bladder.

Pathology.—The biliary ducts are dilated, congested, œdematous, and filled with purulent material, usually bile-stained; and about the terminal branches of the biliary ducts within the liver there are few or many, small or moderately sized, abscesses. The liver tissue is the seat of cloudy swelling and more or less necrosis.

Symptoms.—Usually there is a history of antecedent cholelithiasis or vague symptoms that may be interpreted as due to gallstones; in other cases there is some impairment of the general health, which, in the light of subsequent events, may be ascribed to malignant disease; in still other cases the patient suffers from, or has recently suffered from, some general infection, such as typhoid fever, pneumonia, etc. The onset is often insidious; occasionally the first noteworthy symptom is slight jaundice, but in perhaps most cases, especially in cases in which there has been antecedent jaundice due to other causes, the onset of the disease is announced by constitutional phenomena, such as chills, fever, and sweats. These are usually severe, more or less frequently repeated at irregular intervals, and soon lead to considerable impairment of the general nutrition, emaciation, loss of appetite, nausea, perhaps vomiting, diarrhœa, etc.; variable afebrile periods, however, are not uncommon. Jaundice is almost always present, but it is variable in degree; usually it is slight or moderate only, except in cases in which the cholangitis is associated with other conditions that cause intense jaundice. There may be dull aching or discomfort in the region of the liver (tension of the liver capsule); now and then there is more severe pain due to acute cholecystitis, cholelithiasis, obstruction of the ducts by tumors or worms, perihepatitis, peritonitis, etc. The liver is enlarged and usually continues to enlarge under observation. Usually the gall-bladder also is enlarged and tender (acute cholecystitis). The spleen may be enlarged. Examination of the blood reveals polynuclear leukocytosis, more marked during and just after the febrile periods; sometimes it is absent during the afebrile periods.

Diagnosis.—The diagnosis is usually apparent from a history of antecedent gallstones or other disorder of the biliary tract, or of typhoid fever or other infection, and the development of chills, fever, sweats, jaundice, local pain, progressive enlargement of the liver, perhaps also of the gall-bladder and spleen, and leukocytosis.

Prognosis.—The prognosis is bad, but may be improved by thorough drainage of the gall-bladder and ducts undertaken early.

Treatment.—Attempts should be made to prevent the disorder by thorough and efficient treatment of all forms and grades of biliary infection (including gallstones). When suppurative cholangitis has developed, the treatment is essentially surgical, and consists of the establishment of free drainage. The only available medical treatment comprises the use of antiseptics, such as the salicylates, hexamethylenamin, and other remedies, to disinfect the biliary tract and promote the free flow of bile.

CHRONIC CATARRHAL CHOLANGITIS.

Etiology.—Chronic catarrhal cholangitis may result from repeated attacks of acute catarrh, or from a low grade chronic infection. Usually it is found in association with gallstones; but it may be found also in chronic duodenal catarrh, and in obstruction due to tumors or strictures of the ducts.

Pathology.—The lesions of chronic catarrhal cholangitis (of the larger ducts) consist of congestion and swelling of the mucosa, increased production of mucus, round-cell infiltration, and proliferation of the submucosa and muscularis, with the production of new fibrous connective tissue; these lead to thickening and induration of the walls of the ducts and to pericholangitis. There may be partial or complete obstruction of the ducts. In the event of complete obstruction, the ducts become enormously dilated (up to the size of a thumb or larger); the gall-bladder and some of the intrahepatic ducts also become dilated. The contents of the biliary tract is usually a clear, almost colorless, often sterile mucus; when the mucus is sterile, the mucosa of the biliary tract is usually quite smooth. In the event of incomplete obstruction, the ducts, and especially the gall-bladder, are less dilated, the contents are usually turbid and often contain bacteria, and the mucosa may be more or less ulcerated.

Symptoms.—The symptoms are those of chronic relapsing jaundice. Rarely, this may represent a direct continuation of an acute catarrhal jaundice, being more insidious in onset, and subject to exacerbations and remissions. The attending phenomena are those of acute catarrhal cholangitis—prolonged. In the cases of complete obstruction, the contents of the biliary tract being sterile, there is usually no fever, and remissions in the jaundice are slight. In the cases of incomplete obstruction, remissions in the jaundice are more marked, and the contents of the biliary tract being infected, attacks of chill, fever, and sweats are

common. These attacks by no means warrant a diagnosis of suppuration of the biliary tract, although they are evidences of infection. This chronic catarrhal cholangitis may lead to suppurative cholangitis (of which it is often only a minor manifestation), acute infectious jaundice, acute yellow atrophy of the liver, etc.

Diagnosis.—In cases of persisting jaundice that begin seemingly as acute catarrhal cholangitis, one must attempt to eliminate cholelithiasis and malignant disease obstructing the ducts. Often it is quite impossible to differentiate non-calculous from calculous cholangitis with a gallstone in the common duct; attempts to do so are usually only of academic interest. A history of antecedent gallstone colic is suggestive, but by no means conclusive. Hypertrophic biliary cirrhosis may be eliminated by the absence of marked enlargement of the liver and spleen.

Prognosis.—The prognosis is dependent upon the associated lesions.

Treatment.—The treatment is essentially that of acute catarrhal cholangitis. In protracted cases operation should be undertaken with a view to drain the biliary ducts; this is the more urgently demanded in view of the impossibility often of excluding obstruction due to a gallstone.

ACUTE CHOLECYSTITIS.

Etiology.—It is largely artifice that leads to a distinction between cholangitis and cholecystitis; the one like the other is an expression of infection of the biliary tract. In some cases the infection may remain confined to the ducts or the gall-bladder. In other cases, developing in the one or the other, the process may extend to that not primarily involved; and it may persist in the gall-bladder and subside in the ducts, or *vice versa.*

Acute cholecystitis may result from the microörganisms already mentioned as causative of acute cholangitis. Special importance attaches to Bacillus typhosus, Bacillus coli, and the pyococci. These usually reach the gall-bladder by way of the portal circulation, less commonly by way of the general systemic circulation, and sometimes, perhaps, by way of the diverticulum of Vater. Important contributing factors comprise those that reduce the vitality and resisting power of the gall-bladder and those that interfere with the free flow of bile.

Significance attaches to the fact that the gall-bladder once having become infected, may harbor the invading bacteria for many years (so-called "chronic carriers," especially important in relation to typhoid fever); these bacteria being continuously or intermittently discharged from the gall-bladder, are often the source of epidemics.

Pathology.—Various types of acute cholecystitis may be differentiated —catarrhal, suppurative, phlegmonous, gangrenous, and membranous. Properly interpreted, these are varying manifestations of the one process (an infection), which results from variations in the virulence of the infecting agent, in the local and general resistance, and in the freedom of

the circulation of the bile; they differ in degree rather than in nature, and pass sometimes almost imperceptibly the one into the other. In the mild cases the gall-bladder is distended and tense; the wall is swollen, œdematous, and softened, and the mucosa is, in addition, congested and covered with a layer of mucus, perhaps bile-stained; in many cases the inflammatory phenomena invade also the peritoneal covering, which then is dull, lustreless, or opaque, and covered with a thin layer of fibrin, which may serve to unite the gall-bladder to adjacent structures. The cystic duct usually is partially obstructed from swelling of the mucosa; sometimes it is completely obstructed, but usually only in the event of recurrent attacks of inflammation or of cicatrization of ulcerations. The contents of the gall-bladder usually consist of turbid bile. Gallstones may be encountered, especially in recurrent attacks; but usually not in cases of less than four to six months' duration (during most of which time the infection may be latent). The adjacent lymph nodes, especially those in the portal fissure, are usually enlarged and softened. The more severe cases exhibit all gradations to severe suppurative, ulcerative, and gangrenous lesions, with more or less suppurative contents (acute and chronic empyema of the gall-bladder).

Symptoms.—Initial nausea and vomiting, fever, pain, and tenderness in the region of the gall-bladder, rigidity of the overlying abdominal muscles, and an enlarged and tender gall-bladder constitute the important clinical manifestations of acute cholecystitis. These may be of sudden or rather insidious onset. The nausea and vomiting are usually moderate in grade and soon subside, except in the event of pericholecystic or more general peritonitis and concomitant paresis of the intestine. In catarrhal cholecystitis the fever is slight and lasts only a short time; in the cases complicating some general infection there may be little if any added fever, but if occurring, for instance, during convalescence from typhoid fever, the onset of the cholecystitis is often announced by fever, which may be high. The pain usually occurs first in the epigastrium, sometimes directly in the midline, and later becomes more localized to the right hypochondrium; it varies much in character, and may be colicky. Jaundice is not a part of cholecystitis, but it may occur in the event of complicating cholecystitis, or obstruction of the common duct.

The consequences of acute cholecystitis are many: Persistent mild infection (chronic bacillus carriers) with acute exacerbations; gallstones; pericholecystic adhesions; or progression to severe types of the affection, with ulceration and perforation (sudden increase of the local pain, more or less collapse, increased pulse rate, and increase of the local abdominal symptoms—that is, peritonitis).

Diagnosis.—The diagnosis of acute cholecystitis presents no serious difficulties in the great majority of cases; nausea and vomiting, pain and tenderness in the region of the gall-bladder, rigidity of the overlying abdominal muscles, and an enlarged and tender gall-bladder constitute a complex of symptoms that can scarcely be attributed to anything else. The phenomena of gallstone colic are properly attributable in most cases

to an acute cholecystitis, perhaps acute exacerbation of a chronic cholecystitis. In the severe cases, with well-marked peritonitis of the upper abdomen, one must exclude other causes of such peritonitis, especially perforated gastric or duodenal ulcer, acute pancreatitis, etc.

Prognosis.—In the majority of cases the prognosis is good, the process tending to subside, under appropriate treatment, within ten days or a fortnight. Sequels, especially gallstones and adhesions, however, are almost certain to occur, so that the subsequent outlook is not the best; in other cases a low-grade infection may persist and the patient become a subject of chronic infection and recurring attacks of cholecystitis. In the more severe forms, suppurative, phlegmonous, and gangrenous cholecystitis, the tendency toward spontaneous cure is slight; in this event the prognosis is grave, since not only are the lesions of the gall-bladder serious in themselves, but a localized abscess or general peritonitis may ensue and a fatal issue result, unless recourse is had to surgical intervention.

Treatment.—The treatment is that recommended for gallstone colic (page 735), which virtually is an attack of more or less acute cholecystitis. In the severe, suppurative, and gangrenous cases, surgical intervention is called for.

CHRONIC CHOLECYSTITIS.

Chronic cholecystitis may be the residual manifestations of an acute cholecystitis, or the process may be chronic from the beginning. The etiological factors, therefore, are those of acute cholecystitis, the chronicity of the process being a manifestation of lingering infection, or the consequence of a low-grade infection with almost, but not quite, sufficient biliary drainage. In the great majority of cases the condition is associated with gallstones the one process sustaining the other; that is, most cases are calculous. In a few cases the lesions extend throughout the biliary tract; that is, cholangitis and cholecystitis are associated. In the absence of gallstones, the lesions may be catarrhal and the gall-bladder may be distended. In most long-standing cases the gall-bladder becomes reduced in size, thickened, and sclerotic; it may be represented by a mass of thickened, contracted connective tissue, perhaps enclosing gallstones. Symptomatically, the condition cannot be distinguished with certainty from cholelithiasis.

CHOLELITHIASIS.

(*Gallstones.*)

Etiology.—Cholelithiasis is an exceedingly common condition, being found at the necropsy in from 5 to 10 per cent. of subjects dead from all causes. It occurs at all ages, but the incidence increases progressively with advancing years; 75 per cent. or more of the cases are found in

persons over forty years of age, and less than 1 per cent. in those under twenty years; but this age incidence does not correctly represent the time of the formation of gallstones, since in many cases, having formed, they remain latent for years and are found only when the subject dies at a more advanced age from the consequences of gallstones or other causes. Doubtless, therefore, many, if not most, gallstones are formed in early adult life, before the fortieth year, corresponding with the period of greatest frequency of typhoid and other infections. They are more common in women than in men (the ratio being variously stated as 5 to 1 (Schroeder), to 4 to 3 (Rolleston). The essential cause is a low-grade catarrhal inflammation of the biliary tract, which is usually provoked by the typhoid or colon bacilli, or attenuated pyococci; a contributing factor is some obstruction to the free flow of bile. This ensues in conditions of biliary obstruction, and is promoted by a sedentary life, obesity, muscular inactivity, lax abdominal muscles (as after pregnancy), hepatoptosis, etc. Whether a third factor, disorders of metabolism (disorders dependent upon diet), is active, has not been determined with certainty.

Pathology.—Gallstones may be found in any part of the biliary tract, but they are most common in the gall-bladder. There may be one or many stones; they vary much in size—from the smallest particle (biliary sand or gravel) to those larger than a normal gall-bladder; and they vary in shape. Single stones are usually ovoid, spheroid, or pear-shaped; multiple stones are usually cuboidal or polyhedral, and are faceted (from mutual compression). Stones within the intrahepatic ducts conform to the ducts in shape, and are ovoid or cylindrical; occasionally they are branched, as are the ducts. Although usually smooth, the surface of gallstones may be rough, irregular, or nodular (mulberry calculus). In consequence of the bacterial infection of the biliary tract, a low-grade catarrhal inflammation is set up; this, on the one hand, leads to obstruction of the free flow of bile from swelling of the mucous membrane, and the products of this inflammation, on the other hand, contain the essential constituents of gallstones. In the gall-bladder the catarrhal inflammation leads to desquamation of the lining epithelium, an albuminous exudation, and an increased formation of mucus and of cholesterin; and in the gall-bladder dynamic factors frequently favor the stagnation of bile. The increased cholesterin is derived not from the bile, but from catarrhal disintegration of the mucous cells lining the wall of the gall-bladder. Bacmeister and Exner and Heyrovsky, however, have shown that bacteria, notably the typhoid and the colon bacillus (but not streptococci), may effect directly a decomposition of the bile with the precipitation of cholesterin. Exner and Heyrovsky attribute this to decomposition of the bile acid salts (the solvent menstruum), in consequence of which the normal amount of cholesterin can no longer be held in solution. The bilirubin calcium is derived from the bile, being precipitated by bacterial growth or by the albuminous exudation of the inflammatory process. Bilirubin calcium is believed by Naunyn to act as a cement substance, binding together the cholesterin, desquamated

epithelium, etc., the whole forming the nucleus of the gallstone. Agglutinated typhoid and other bacteria also may apparently act as the nucleus.

The following varieties of gallstones are distinguished by Naunyn: (1) Pure cholesterin stones, which consist of nearly pure cholesterin, and are uncommon. They vary in size from that of a cherry to that of a pigeon's egg, are hard, oval, or roughly spherical, seldom faceted, have a smooth or nodular surface, and are pure white or yellowish and translucent, or rarely brown, greenish, of brownish-black on the surface. On section they are white, and crystalline throughout, or show brown deposits between the crystals; they are not stratified. (2) Laminated cholesterin stones, which consist of about 90 per cent. of cholesterin, together with bilirubin calcium, biliverdin calcium, and calcium carbonate. In general appearance they resemble the pure cholesterin calculi, but they may be brittle and friable, and they are often faceted. On section, they are more or less distinctly laminated, layers of almost pure white alternating with others that are yellow, brown, green, or red. The centre may be crystalline, but the external layers are usually vitreous or earthy. (3) The common (or mixed cholesterin) stones, which vary much in size, although they seldom equal a large cherry; they may be small and very numerous, and are usually faceted. The surface is usually yellow, but often brown or white. When fresh, they are often soft and greasy; when dried, they undergo shrinkage and become harder. On section, the centre is often soft and may contain a cavity. (4) Mixed bilirubin calcium stones, which may consist roughly of 75 per cent. of bilirubin calcium and about 25 per cent. of cholesterin. They are as large as a cherry, or larger, and may occur singly or in groups of three or four in the gall-bladder or larger ducts. Their shape depends upon their situation; when multiple, they may be faceted. They consist of concentric layers of reddish-brown or dark brown material, which is seldom quite hard, and contracts on drying, often with the formation of fissures or cracks. (5) Pure bilirubin calcium stones, which vary in size from that of a grain of sand to that of a pea. There are two types: small, solid, brownish-black concretions, with rough, irregular surfaces and wax-like consistency, and exhibiting a disposition to become welded; and harder, smooth concretions of a grayish-black metallic lustre and an internal spongy structure. (6) Rarer stones, such as amorphous and incompletely crystalline cholesterin gravel (resembling pearls); calcareous stones; concretions with included bodies and conglomerate stones; and casts of the bile ducts.

Symptoms.—The characteristic and significant symptoms are: (1) Chronic, long-continued, or recurring indigestion, and (2) the phenomena designated gallstone colic. The symptoms of chronic long-continued or recurring indigestion are of the utmost importance and are commonly misinterpreted. This stands in relationship with the facts—that gallstones are present in very many subjects and are commonly believed not to cause noteworthy symptoms (in not more than 5 per cent. of the subjects); that symptoms, having once occurred, are exceedingly likely to recur; and that the symptoms are often paroxysmal and very severe.

Gallstones are sometimes latent, particularly in elderly subjects, in whom the original infection has died out, and in whom more or less atrophy, especially of the muscular coat of the gall-bladder and biliary ducts, has occurred; but there can be no doubt that symptoms commonly denominated "stomach trouble," "indigestion," "dyspepsia," etc., are often due to gallstones and disease of and about the biliary tract.

Gallstone colic usually develops suddenly with severe, often agonizing, pain in the right hypochondrium or the epigastrium, radiating around the chest or to the right scapular region, nausea, vomiting, and prostration—weak, rapid pulse, rapid heart action, profuse perspiration, etc. Sometimes the attack is preceded by local, less acute pain or discomfort, chilliness, fever, etc.; more commonly (60 per cent. or more of the cases) chilliness and fever accompany and follow the pain. In some cases the severe pain lasts for a few moments only; usually it lasts from two to twelve hours; in unusual cases it may last much longer, but in these circumstances the phenomena are more correctly interpreted as a succession of paroxysms. Often the pain ceases as suddenly as it began, but usually following the agonizing pain, more or less dull aching continues for a variable period. The colic may recur at varying intervals—days, weeks, months, or years; occasionally there is only one attack; usually there are many. With succeeding attacks the likelihood of other attacks increases. In some cases the pains are not colicky; that is, the classical gallstone colic does not occur. The pains, of whatever nature, are usually due to an acute cholecystitis, or reactivation of a more or less quiescent cholecystitis; but, as pointed out by Riedel, the colicky pains may be due to: (1) Adhesions of a gall-bladder no longer containing stones; (2) adhesions when large stones are present in the gall-bladder and the cystic duct is patent; (3) inflammatory processes in a gall-bladder distended by fluid or stones, when the cystic duct is occluded by inflammation or by the presence of a stone in the neck of the gall-bladder; (4) the transit of a stone through the bile passages; and (5) the inflammation of a dilated, calculous common duct or its tributaries, without impaction of a stone.

Jaundice may occur, but is entirely absent in at least one-third of the cases. It varies much in degree and duration with the local conditions. It may be due to: (1) Gallstones in the hepatic or the common bile duct; (2) spasm of the musculature or inflammatory swelling of the mucosa of the biliary ducts, and (3) compression of the common duct or of the extrahepatic part of the hepatic duct by a large stone in the cystic duct, swollen lymph nodes, regional tumors, inflammatory exudations, adhesions, kinking of the ducts, etc. The gall-bladder may be enlarged and palpable in early cases; but in advanced cases, when the gall-bladder has become thickened and cicatrized and is manifestly incapable of distending, it is usually not palpable. Courvoisier stated that in cases of chronic jaundice, contraction of the gall-bladder is suggestive of gallstones, and that dilatation of the gall-bladder is suggestive of biliary obstruction, caused by factors other than gallstones; that is, pressure

from without (most often enlargement, carcinoma, of the head of the pancreas). This, so-called Courvoisier's law, is applicable in perhaps two-thirds of the chronic cases.

Fever is common, and should be correctly interpreted—as a manifestation of infection, and not as due to reflex causes, etc. In many cases, concurrently with the development of the gallstone colic, there is a sudden elevation of the temperature, followed sometimes by an equally sudden fall; in other cases, the fever is of longer duration—obviously due to acute cholecystitis, or an acute exacerbation of a chronic cholecystitis; in still other cases, especially cases of calculus in the common duct, the fever is that known as Charcot's intermittent hepatic (or biliary) fever—periodic attacks of chill, fever (103° to 104°F.), and sweats, usually accompanied by pain in the epigastrium or right hypochondrium, recurrence or increase of jaundice, perhaps nausea and vomiting, and often leukocytosis.

Obstruction of the Cystic Duct.—The calculus is usually lodged in the beginning of the duct or in the termination of the neck of the gall-bladder. The following conditions may ensue: (1) Acute cholecystitis (catarrhal, suppurative, phlegmonous, or gangrenous). (2) Chronic dilatation of the gall-bladder, a condition that follows complete obstruction to the entrance of bile into the gall-bladder, which soon becomes distended with mucus, at first bile-stained, but ultimately quite clear (so-called hydrops). (3) Chronic fibrous or atrophic cholecystitis—the usual sequel of hydrops when the obstruction is not complete and the infection dies out. The gall-bladder gradually lessens in size and may ultimately be reduced to a mass of fibrous tissue, which usually tightly encircles a calculus.

Obstruction of the Common Bile Duct.—The obstruction may be complete or incomplete, and may or may not be associated with cholangitis. When the obstruction is complete, a stone is usually tightly impacted in the common duct; when the obstruction is incomplete, the stone may be more or less fixed, but permits the flow of bile around it, or it may circulate in the common duct and imitate the conditions in a ball valve. Dilatation of the biliary ducts usually ensues, and to the common cholangitis and pericholangitis more or less fibrosis of the adjacent liver tissue and atrophy of the liver cells often ensue—a condition sometimes spoken of as *obstructive biliary cirrhosis* In the event of complete obstruction without infection, deep and permanent jaundice is produced, and is unattended by fever. Incomplete obstruction may last for many years without leading to jaundice. Usually, however, jaundice is present; it lasts a long time, varies in intensity with the degree of obstruction, is subject to more or less fluctuation, which is very characteristic; and there is fever, periodic, intermittent, or remittent, or more or less continuous (indicative of the usually associated biliary infection). Osler has emphasized the importance of the following signs in the diagnosis of a ball-valve calculus in the diverticulum of Vater (where it occurs most frequently), or in the common bile

duct: "(*a*) Ague-like paroxysms of chill, fever, and sweating—the hepatic intermittent fever of Charcot; (*b*) jaundice of varying intensity, which persists for months or even years, and deepens after each paroxysm; and (*c*) at the time of the paroxysm, pain in the region of the liver, with gastric disturbance. . . . Pain, which is sometimes severe and colicky, does not always occur. There may be vomiting and nausea. As a rule, there is no progressive deterioration of health. In the intervals between the attacks the temperature is normal."

Complications and Sequels.—Of the many complications and sequels of cholelithiasis, some infectious, some purely mechanical, but most of them infectious and mechanical, the following are the most important: (1) *Infectious (Inflammatory) Lesions of the Biliary Tract and Adjacent Viscera.* These comprise especially varying grades of acute and chronic, suppurative and non-suppurative, cholangitis and cholecystitis, progressing perhaps to ulceration, gangrene, perforation, etc. The inflammatory phenomena may extend to adjacent structures, and set up local peritonitis (with abscess formation) or a more widespread peritonitis, pylephlebitis, pancreatitis, etc. (2) *Chronic Pericholecystic Adhesions.* These represent the sequels of postsubacute and chronic, sometimes acute, inflammation that has spread to the adjacent peritoneum. Similar adhesions may follow disease of the stomach and duodenum; and any of these adhesions may be responsible for "stomach ache," "indigestion," "biliousness," etc. (3) *Biliary Fistulæ.* Fistulous communications between the biliary tracts and adjacent viscera, as well as the exterior of the body, frequently result from cholelithiasis and the associated inflammatory and ulcerative processes. Courvoisier in a study of 499 cases, found that fistulous communications had been established as follows: between different parts of the biliary passages in 8; into the portal vein in 5; into the peritoneal cavity in 70; into peritoneal adhesions in 49; into the retroperitoneum in 3; with the stomach in 13; with the duodenum in 83; with the jejunum in 1; with the ileum in 1; with the colon in 39; with the urinary tract in 7; with the pleura and lung in 24; and externally in 196. (4) *Intestinal Obstruction.* This is a rare event, that may follow ulceration of a gallstone from the gall-bladder into the duodenum, or, less commonly, the colon. The obstruction occurs most commonly in the ileum (near the ileocecal valve), and may be suspected in a person in advanced life with a history of past gallstones.

Diagnosis.—The diagnosis of cholelithiasis comprises the recognition of the gallstone colic and of the many and varied associated lesions and sequels. Severe pain in the epigastrium or the right hypochondrium, radiating around the chest or to the right scapula, accompanied by nausea and vomiting and enlargement and tenderness of the gall-bladder and followed by jaundice and the detection of a gallstone in the feces, are characteristic of the passage of a gallstone; but jaundice is by no means always present, and the other symptoms may be provoked by cholecystitis in the absence of a gallstone; indeed, with increasing experience, it becomes often more and more difficult to say whether or not

gallstones are present in certain individual cases of obvious chronic biliary infections. Recourse to the *x*-rays is seldom of much diagnostic utility, since the cholesterin stones produce scarcely any shadow, usually not more than the adjacent liver; in the rare cases of calcium stones, more shadow is cast, but this also is obscured by the shadow of the liver.

The severe pain of gallstone colic may be imitated by renal colic or intestinal colic; but in renal colic the pain is usually in the back, loin, or groin, and radiates down the ureter to the testicle, which may become retracted; strangury or frequent micturition is common; the kidney is usually tender (the tenderness being in the loin rather than anteriorly in the region of the gall-bladder); and the urine subsequently contains blood and a proportionate amount of albumin, perhaps crystals or a calculus, and pus and epithelium from the pelvis of the kidney; and it does not contain bile. Intestinal colic is usually due to some more or less obvious cause; the pain is usually about the umbilicus; it radiates in different directions, remits and intermits, and subsides with the removal of the exciting cause, often with belching or the discharge of feces or gas or mucus by the bowel; and there is no localized tenderness in the region of the gall-bladder. In lead colic there is, in addition, the history of exposure to the poison, a blue line on the gums, anemia, basophilic degeneration of the erythrocytes, and perhaps other present or past evidences of the intoxication, such as persistent abdominal discomfort or pain and constipation, wrist-drop, etc. When the symptoms, especially the pain, are less acute, and do not merit the designation gallstone colic, difficulty may be experienced in differentiating disorders of the stomach, especially gastric and duodenal ulcer. The persistence of jaundice may suggest malignant disease of the ducts or of the pancreas, but in cholelithiasis there is less impairment of the general health; there is usually periodic variation in the intensity of the jaundice (of much diagnostic importance); and fever, especially intermittent hepatic fever, is likely to be present.

Prognosis.—The prognosis, as a rule, depends more upon the associated conditions than upon the mere presence of the gallstones. The prognosis in gallstone colic is virtually that of a more or less acute cholecystitis; the outlook as regards permanent health depends upon the chronic gall-bladder infection. A first gallstone colic is sometimes the last and the infection subsides. Usually, however, there are subsequent attacks, if not of severe colic, of more or less disturbance with digestion, and general impairment of health due to the toxic absorption, etc.; some subjects pass into chronic invalidism from unrecognized chronic biliary infections. The occurrence of intermittent hepatic fever, especially of severe ague-like paroxysms, renders the prognosis bad, since it shows active infection, which, although it may persist for years without suppuration, may nevertheless lead to serious consequences. The prognosis as to final recovery of health is bad without operative intervention in pericholecystic adhesions, fistulæ, and intestinal obstructions.

Treatment.—The fact that gallstones are most common in women, in the obese, in those that lead a sedentary life, eat too much, are constipated, and addicted to alcohol, suggests the correction of certain very obvious etiological factors. Women who have been repeatedly pregnant and have lax abdominal walls should wear proper supports. Men and women who lead a sedentary life should take properly regulated exercise; they should practise deep breathing (to promote the movements of the diaphragm), bathe frequently, be much in the open air, etc.; that is, they should apply to themselves the general principles of hygienic living. Constipation should be overcome, and the diet should be such as will be presently pointed out.

When gallstones have formed, the main indications for treatment may be said to be: (1) To cause solution of the stone or stones; (2) to cause the discharge of the stone or stones; and (3) to treat the complicating infection.

It is quite impossible to cause solution of gallstones in the gall-bladder, olive and other oils, turpentine, ether, etc., being without effect in this regard. The many stones said to have been passed by the bowel after the administration of olive oil are merely masses of saponified oleic acid, resembling gallstones in outward appearance. Olive oil, however, is often of service in relieving the commonly attendant hyperchlorhydria. To attempt the discharge of gallstones from the gall-bladder, even were this possible to effect, is not a worthy object of rational treatment. The special function of the physician is to treat the associated biliary infection.

The *pain of gallstone colic* is usually so severe as to require the administration of morphine, which not only relieves the pain (and mitigates the attendant shock), but, by relaxing the spasm of the biliary musculature, favors the exit of bile from the tense gall-bladder and the transit of the calculus through the ducts (if it be already therein). Usually, $\frac{1}{4}$ grain (0.015 gram) with $\frac{1}{50}$ grain (0.0013 gram) of atropine, or $\frac{1}{100}$ (0.0006 gram) grain of nitroglycerin suffices; sometimes after the lapse of fifteen to thirty minutes a second smaller dose may seem necessary, but in these circumstances a few whiffs of chloroform, or spirit of chloroform or of ether (1 dram, 4 c.c.), to which tincture of belladonna (5 to 10 minims, 0.3 to 0.6 c.c..), may be added, will usually carry the patient through the painful period until the morphine has had time to act; or, and this is often attended by excellent results, an additional dose of atropine ($\frac{1}{100}$ grain, 0.0006 gram) may be administered hypodermically. Should resort be had to chloroform inhalation, one should not induce complete anesthesia in the hope of thus relaxing the biliary musculature and freeing the calculus; such objects are not thus achieved. In many cases relief may also be afforded by hot fomentations over the region of the gall-bladder and by lavage of the stomach with water as hot as can well be borne. Should the vomiting persist and be manifest especially by ineffectual retching, resort should be had to lavage, if it has not been previously used; sodium bicarbonate may be added to the hot water. The hot full bath may also be resorted to at the very beginning of the attack, with

hope of excellent result. In other cases the vomiting may be relieved by bismuth, carbolic acid, creosote, dilute hydrocyanic acid, cerium oxalate, sips of hot water and brandy, champagne, etc. In some cases the pain leads to extraordinary depression of the patient, which, if it is not relieved by the morphine, should be met with whisky or brandy in hot water, or the hypodermic use of camphor.

Upon the subsidence of the pain, as well as in the absence of a definite gallstone colic, the medical treatment of cholelithiasis is that of the chronic biliary infection. The diet should be regulated; large amounts of an alkaline water should be taken, and the saline cathartics should be administered daily. Salicylic acid seems to augment the flow of bile by stimulating its production, and since it also possesses antiseptic properties and is excreted with the bile, it exerts a most desirable influence on the biliary infection. No preparation is better than chemically pure salicylic acid; it may be combined with an equal amount of sodium bicarbonate or sodium benzoate; or the salicylates or other preparations of salicylic acid may be given.

Hexamethylenamin also is excreted in the bile and through the wall of the gall-bladder, and serves to disinfect the biliary passages. It should be given in generous dose—40 to 60 grains (2.5 to 4 grams)—within twenty-four hours, from time to time; but its general effect should be carefully observed. Oxgall also stimulates the secretion and outpouring of bile, and thus favors drainage of the biliary tract; it should be administered as salts of the bile acids, such as sodium glycocholate, 10 to 15 grains (0.6 to 1 gram) daily. Ammonium chloride also is sometimes extolled for its effect upon inflamed mucous membranes; by promoting the free flow of thin bile it may assist in the much desired flushing of the biliary tract.

Surgical treatment is indicated in all cases in which quiescence or latency of the infection cannot be brought about by medicinal measures; in the event of recurring attacks of gallstone colic; in the cases that run their course under the guise of "indigestion," "biliousness," etc., and in which recurrence of attacks is common; in cases of persisting jaundice, with or without Charcot's biliary intermittent fever; and in all cases of complicating or severe suppurative, ulcerative, or gangrenous cholecystitis.

CARCINOMA OF THE GALL-BLADDER AND BILIARY DUCTS.

Carcinoma may develop in any part of the biliary tract, but it is most common in the gall-bladder. It is usually associated with gallstones, commonly involves the fundus, and may be a fungus-like or an infiltrating growth (cylindrical or cuboidal cell). Extension to the liver occurs in more than 50 per cent. of the cases. In the biliary ducts carcinoma is most common in the common duct, but may occur anywhere, even in the ampulla of Vater (choledochopancreatic duct); it is usually an infiltrating growth.

Symptoms.—The symptoms of carcinoma of the gall-bladder consist usually of antecedent phenomena referable to cholecystitis and cholelithiasis, to which become added a progressive, irregular, and nodular enlargement of the gall-bladder (more than one-half of the cases), jaundice (three-fourths of the cases), and impairment of the general health—emaciation, anemia, and cachexia. Ascites occurs in about one-fourth of the cases, being due to portal obstruction or perihepatitis. In carcinoma of the extrahepatic biliary ducts, jaundice is usually the first noteworthy symptom; it is progressive, soon becomes extreme, and is permanent. The stools become acholic. The gall-bladder becomes enlarged (smooth, pear-shaped), and metastasis may occur to the liver (palpable nodules), to the portal lymph nodes (causing ascites), etc.; and the patient becomes cachectic.

Prognosis.—Death usually occurs within six months of the onset of serious symptoms.

Treatment.—The treatment is essentially surgical.

DISEASES OF THE PANCREAS.

Pathological Physiology.—Some reference has been made in the introductory remarks on diseases of the intestine to the pathological physiology of the pancreatic juice, and, under diabetes, to disturbances in the internal secretion of the pancreas. The chief results of disturbances (deficiency) of the pancreatic juice consist of imperfect tryptic digestion —the occurrence of unchanged muscle fibers in the stools (azotorrhœa), especially the non-digestion of the muscle fiber nuclei (Schmidt's test); imperfect digestion of carbohydrates, due to lessening or absence of pancreatic amylase; and imperfect fat-splitting, due to lessening or absence of steapsin. The stools are voluminous; pale, grayish, slate-colored, or clay-colored; pasty, fetid, and acid. The fat content may be as high as 80 or 90 per cent. (steatorrhœa), instead of the normal 7 to 10 per cent. In addition, the urine may contain sugar, rarely a fat-splitting ferment (Opie), or it may reveal the so-called Cammidge reaction.

HEMORRHAGES INTO THE PANCREAS.

Hemorrhage into the pancreas occurs in a variety of conditions such as determine hemorrhage in other organs—infectious diseases, sepsis, hemorrhagic diathesis, purpura, in association with tumors and cysts, and following traumas. In addition, however, there is a class of cases in which hemorrhage into the pancreas constitutes the only noteworthy lesion at necropsy in cases of sudden death occurring in persons previously healthy—so-called pancreatic apoplexy. Many of these are doubtless cases of hemorrhagic pancreatitis, being reported in the older literature, when observations were more or less imperfect and the nature

of hemorrhagic pancreatitis ill understood. From these, however, may be separated cases occurring in atheroma, and in association with thrombosis and embolism. Symptomatically, however, they cannot be differentiated from acute or hemorrhagic pancreatitis.

ACUTE PANCREATITIS.

Etiology.—Acute pancreatitis is most common in adult males, although it occurs in women and has been observed even in infants. Some of the patients have given a history of addiction to alcohol; others have suffered from gastro-intestinal derangements, cholelithiasis, or diabetes mellitus. In most cases the disease is associated with cholelithiasis and concomitant infection of the biliary tract. Usually the gallstone obstructs the common bile duct, and thus permits the ready access of the bile to the pancreas. Flexner, Pearce, and others have demonstrated that the injecting of bile, artificial gastric juice, and other irritating substances, into the pancreatic duct may cause pancreatitis, even in the absence of bacterial infection. In some cases ulcerative and suppurative processes of adjacent organs or of remote organs (by metastasis) may set up the pancreatitis.

Pathology.—Since the time of Fitz's original observations it has been customary to distinguish three varieties of acute pancreatitis—hemorrhagic, suppurative, and gangrenous. In many cases, however, it is impossible to differentiate between so-called pancreatic hemorrhage, hemorrhagic pancreatitis, and gangrenous pancreatitis, and in most cases these are but different stages or varying manifestations of the one disease. In *hemorrhagic pancreatitis* the pancreas is enlarged, softened, and mottled, dark reddish brown or black in color—the result of infiltration of the interstitial tissue with blood. In some cases the hemorrhagic infiltration extends also to the neighboring peripancreatic tissues, and the mesentery, mesocolon, and omentum. An essential feature of the disease is extensive necrosis of all the constituents of the gland—the parenchyma, the interstitial tissue, and the bloodvessels, with which inflammatory phenomena (polynuclear leukocytic infiltration and serofibrinous exudation) are associated. The hemorrhage is attributed to necrosis and consequent rupture of the bloodvessels. The necrotic foci are sharply demarcated from the surrounding living tissue by a narrow zone of reactive inflammation. Opie draws attention to the occurrence of hyaline thromboses within the relatively intact tissue adjoining the necrotic zones, and he believes that "it is not improbable that the ferments of the pancreatic juice, notably trypsin, acting upon cells injured by contact with various irritant substances which can produce hemorrhagic pancreatitis, have a part in the production of the widespread disintegration of the parenchyma which results."

Gangrenous pancreatitis follows hemorrhagic pancreatitis, and no sharp line of demarcation can be drawn between them. The initial lesions are similar to those of hemorrhagic pancreatitis—necrosis and

consequent hemorrhage; inflammatory alterations soon develop, and in the course of time, concurrent with bacterial invasion, the appearances recognized as gangrene result. The pancreas is swollen, soft, friable, and mottled grayish-black (slate-colored) or reddish brown or black in color. In some cases it consists of a dark, mottled, slate-colored, foul-smelling necrotic mass; whereas, in a few cases, late in the course of the disease, it may consist of a rather firm and dry mass covered with blood. Inflammation of the lesser peritoneal cavity results, and is often followed by the development of a large abscess cavity containing dark, grumous, malodorous, chocolate-colored, purulent material, and in some cases the partly or fully sequestered gangrenous pancreas. Rupture of the abscess into the intestine may ensue.

Suppurative pancreatitis is a secondary condition, and not the cause of the gangrene. There may be a single large abscess or many small abscesses—the pancreas being correspondingly enlarged and softened. The suppuration may extend to the adjacent tissues, or rupture may occur into the lesser peritoneal cavity (giving rise to a large abscess cavity), or into the stomach or the duodenum; or pylephlebitis and liver abscess may result.

Fat necrosis is a common concomitant of acute hemorrhagic and gangrenous pancreatitis, and may be observed also in association with chronic suppurative and chronic interstitial pancreatitis. It occurs as disseminated or multiple minute foci in or upon the pancreas, and as larger foci (2 to 3 mm. in diameter) in the fat of the omentum, and of the subperitoneal and retroperitoneal tissues, and sometimes in the pericardial and subcutaneous fat. Extremely large foci have occasionally been observed. The foci are whitish or yellowish and opaque (in marked contrast to the translucent yellow of the normal fat), and they are frequently surrounded by a narrow zone of hemorrhage. They consist of lime and certain fatty acids. Flexner has demonstrated the presence of the fat-splitting ferment (presumably that of the pancreatic juice) in distant necrotic foci (subcutaneous and peritoneal fat), as well as in the foci in the immediate vicinity of the pancreas. Fat necrosis is believed to result from diffusion of the pancreatic ferment in consequence of hemorrhagic destruction of the gland or obstruction of the ducts.

Symptoms.—As a rule, the patient presents a history of long-standing gastro-intestinal derangement; sometimes of more or less distinct and recurring attacks of colicky pain, nausea, and vomiting, attributable in some cases to cholelithiasis, in others, perhaps, to minor attacks of pancreatitis from which the patient has recovered. Acute pancreatitis itself is characteristically acute in onset, with sudden, severe, continuous pain in the epigastrium, nausea, and vomiting, soon followed by collapse, subnormal temperature, rapid pulse, and sometimes death within twenty-four to forty-eight hours. The pain, though continuous, is subject to paroxysmal exacerbations, and is intensified by motion and vomiting; as a rule, it is not very well localized—spreading from the epigastrium over the upper part of the abdomen (doubtless due to stretching and

tearing of the cœliac plexus). Occasionally the pain is more marked to the left of the median line—when the lesions are more marked in the tail of the pancreas. The epigastrium is exquisitely tender, especially on deep pressure. Should the patient not die, evidences of peritonitis of the upper part of the abdomen soon develop. The pain and tenderness continue; rigidity of the abdominal muscles becomes marked; and distention, due to paresis of the stomach and intestine, ensues. The bowels are markedly constipated (whence the condition may be mistaken for intestinal obstruction), the temperature rises and may reach 104° F. (though this is unusual), the pulse becomes rapid and thready, hiccough and thirst may increase the distress of the patient, the features become pinched and pallid, the expression anxious, and some mental wandering and delirium may supervene. Physical examination may disclose a deep-seated swelling or merely induration—tympanitic on percussion because of the overlying stomach and intestine. A chill sometimes occurs at the onset, but is more common and is likely to be repeated later, and to be associated with sweating and leukocytosis, when suppuration has supervened. Slight jaundice is observed in some cases, and is attributed to obstruction of the common bile duct or this and the pancreatic duct by a gallstone in the diverticulum of Vater, by swelling of the duodenal mucous membrane, by the enlarged and swollen pancreas, or by an abscess which may develop. The urine is lessened in amount, and may contain albumin and casts, but rarely sugar. Opie has suggested that the fat-splitting ferment may be found in the urine in some cases and recommends this as a test of possible diagnostic value. An abscess may rupture into the intestine, and blood and pus, and even a portion of the necrotic pancreas, may appear in the stools. If the disease is not fatal within several days, the acute symptoms subside, and rarely the patient may eventually recover or the disease may become chronic. Usually, however, if not relieved by operation, the patient dies from exhaustion and sepsis in the course of several days or weeks.

Diagnosis.—"Acute pancreatitis is to be suspected when a previously healthy person or a sufferer from occasional attacks of indigestion is suddenly seized with a violent pain in the epigastrium followed by vomiting and collapse, and in the course of twenty-four hours by a circumscribed swelling, tympanitic or resistant, with slight elevation of temperature. Circumscribed tenderness in the course of the pancreas and tender spots throughout the abdomen are valuable diagnostic signs" (Fitz). The different forms of acute pancreatitis cannot be differentiated the one from the other, though suppuration is suggested by the ordinary evidences of sepsis. Other causes of peritonitis in the upper part of the abdomen should be taken into consideration, but it is often impossible to differentiate before operation, perforation of a gastric or a duodenal ulcer, though the history sometimes affords a clue. In cholecystitis the pain is less severe, is located over the gall-bladder, and radiates to the right and the back; muscular rigidity is less widespread; and tympany, if it occurs, is delayed and less marked. A history of previous biliary

colic and jaundice is inconclusive, since it may be present in pancreatitis as well as in cholecystitis. Irritant (and ptomaine) poisoning is suggested by the history of the case, analysis of the vomit, generalized abdominal pain, and diarrhœa (as contrasted with constipation).

Treatment.—The nature of the disease precludes the possibility of medicinal treatment being of much avail. At the onset, attempts should be made to relieve the pain by hot fomentations locally and morphine hypodermicly, and to counteract the collapse by stimulants and external heat. The treatment of the disease itself is essentially surgical—exploration and drainage of the lesser peritoneal cavity. In some cases this may be undertaken with hope of ultimate success, if the patient survives the initial shock. The supervention of suppuration, of course, calls for operative intervention.

CHRONIC PANCREATITIS.

Etiology.—Chronic interstitial pancreatitis (fibrosis of the pancreas) is more common in men than in women, especially between the fortieth and the sixtieth year (two-thirds of the cases). Its etiological factors are: (1) Bacterial infections, the consequence of ascending infection of the ducts from the duodenum, or of infection transported by the bloodvessels or the lymphatics, as in syphilis, tuberculosis, etc. (2) Intoxications, especially alcohol, probably answerable for the rather common association of interstitial lesions in the pancreas and cirrhosis of the liver. (3) Mechanical factors. Opie, who has studied the subject attentively, states that "the most common cause of chronic pancreatitis is obstruction of the duct of Wirsung, due to pancreatic calculi, to biliary calculi in the terminal part of the common bile duct, or to carcinoma invading the head or body of the gland. Obstruction of the duct may be followed by the invasion of bacteria, and these take part in the production of the resulting lesion. Ascending infection of the unobstructed duct of Wirsung may follow an acute lesion of the duodenum or of the bile passages, and may cause chronic inflammation. In cases which give a history of long-standing vomiting, chronic diffuse pancreatitis may be found at necropsy, and is probably the result of an ascending infection of the gland."

Pathology.—The noteworthy lesion consists of enlargement and induration of the head of the pancreas, in some cases of the entire organ. Opie distinguishes two varieties of chronic pancreatitis: (1) Interlobular pancreatitis, the result of obstruction of the ducts, and sometimes of ascending infection, and characterized by proliferation of connective tissue between the lobules, only secondary invasion of the lobules, and non-implication of the islands of Langerhans, except in a very advanced stage of the disease; and (2) interacinar (or intralobular) pancreatitis, much less common than interlobular pancreatitis, and characterized by proliferation of the interacinar connective

tissue with early invasion and hyaline degeneration of the islands of Langerhans. This accompanies atrophic or Laënnec's cirrhosis of the liver, arteriosclerosis, and hemochromatosis. A congenital syphilitic pancreatitis, characterized by diffuse interstitial connective-tissue hyperplasia and atrophy of the parenchyma, but with preservation of the islands of Langerhans, is also described.

Symptomatology.—The symptoms of chronic pancreatitis are quite obscure, and in many cases the condition is merely an interesting finding at the necropsy. Occasionally, however, local symptoms due to the situation of the organ, evidences of interference with the motility of the stomach and duodenum, and disturbances with the functions (the internal and the external secretions) of the gland, permit an inference as to the presence of chronic inflammatory alterations. The symptoms develop insidiously, rarely following an acute attack, and consist of gradually increasing dyspepsia, loss of appetite, nausea, flatulence, and epigastric distention. Deep-seated pain, tending to radiate to the left and the back, is occasionally complained of, and on palpation, deep-seated tenderness and sometimes a sensation of increased resistance, corresponding to the pancreas, may be elicited. Vomiting sometimes results from motor insufficiency and dilatation of the stomach, induced by enlargement and induration of the head of the pancreas; occasionally it is frequent and persistent. Jaundice, which may be slight or marked and prolonged, is common also in conditions of enlargement and induration of the head of the pancreas (as well as in tumor formations), and results from obstruction of the common bile duct. Some cases of so-called catarrhal jaundice have been attributed by Mayo Robson to enlargement of the head of the pancreas. Obstruction of the portal vein may lead to the development of ascites. Glycosuria and other evidences of diabetes mellitus occur in a few cases—especially when the islands of Langerhans are destroyed; and since this may happen without much involvement of the glandular structure of the organ, disturbances of pancreatic digestion in the intestine may be absent in the presence of glycosuria. Alimentary glycosuria has been observed in many cases. The stools are voluminous and contain undigested muscle fibers (azotorrhœa) and visible fat (steatorrhœa), but azotorrhœa is found also in conditions, such as obstruction of the ducts, from a tumor, a cyst, or a calculus, interfering with the flow of the pancreatic juice into the intestine; and steatorrhœa occurs in obstruction of the bile duct as well as the pancreatic duct, and after the ingestion of large quantities of fat. The chemical constitution of the fat, however, may be of some diagnostic value, since the investigations of Müller seem to show that normally (as well as in conditions causing deficiency or absence of bile from the intestine) 75 per cent. of the fat in the feces is split up into fatty acids and soaps, whereas in disease of the pancreas (causing deficiency or absence of the pancreatic juice in the intestine) the fat is present largely as free neutral fat. It has been suggested that in such cases a useful test of the presence or absence of the pancreatic juice might result from the giving of pan-

creatic preparations with the subsequent disappearance from the stools of unchanged muscle fibers and fat. However, a marked difficulty in the diagnosis of pancreatic disorders consists in the fact that the functions of the pancreas are shared by other organs. Diarrhœa is rather common; secondary anemia and emaciation are features of some cases; the skin may become pigmented; and profuse salivation (pancreatic sialorrhœa) occasionally occurs and may become distressing.

Diagnosis.—The diagnosis is always difficult, and frequently is scarcely more than a surmise. The most trustworthy symptoms consist of chronic indigestion, more or less jaundice, a disposition to diarrhœa and large stools, and emaciation. Carcinoma of the pancreas, carcinoma of the bile ducts, a calculus in the common bile duct, and perigastric adhesions (following gastric ulcer or carcinoma), must be considered in the differential diagnosis.

Prognosis.—The outlook is good in the event of surgical intervention.

Treatment.—The treatment, aside from operative intervention, is largely hygienic and dietetic. Diastatic ferments, or pancreatic preparations, are sometimes of service in aiding digestion. The causative factors should receive attention in all cases. Of these, gallstones are of prime importance—whence the good effects of operative intervention (drainage of the biliary ducts as a preventive, as well as a curative measure).

CYSTS OF THE PANCREAS.

Cysts of the pancreas are about equally common in the two sexes—of 121 of the 134 cases collected by Oser, 60 were in men, and 61 in women. Sixty-two of the cases (51 per cent.) occurred between the ages of twenty and forty years, but the greatest number (21 cases) occurred in men during the fourth decade, and the greatest number in women (21 cases) during the third decade. The condition, however, has been observed in infancy. Korte distinguishes three varieties: (1) Traumatic cysts, following blows and oft-repeated pressure on the abdomen; (2) postinflammatory cysts; and (3) cysts of unknown origin. Mayo Robson distinguishes anatomically: (1) Retention cysts; (2) proliferation cysts—cystic adenoma and epithelioma; (3) hydatid cysts; (4) congenital cysts; (5) hemorrhagic cysts; and (6) pseudocysts. The cyst may develop from any part of the pancreas, though probably most frequently from the tail, and it varies much in size and in situation in different cases. It may be single or multiple, and microscopic in size or so large as to accommodate many liters of fluid (a cyst containing 14 liters has been reported). The fluid contents is usually slightly opaque and straw-colored, though in traumatic cases it may be hemorrhagic. Its reaction is alkaline; its specific gravity varies between 1007 and 1028 (Oser); and microscopicly it contains fat granules, cell detritus, blood-coloring matter, and sometimes cholesterin. Occasionally, but often not in the older cases, the fluid digests albumin and fibrin (important), and emulsifies fat and

converts starch into glucose (of relatively little importance, since other fluids of the body sometimes possess the same qualities). Most cysts develop in the lesser peritoneal cavity, displace the stomach upward and to the right, and present between the stomach and the transverse colon; in some cases the cyst presents above the stomach, which it displaces downward; occasionally it develops in the transverse mesocolon and presents below the colon; and in still rarer cases it presents toward the splenic or the nephritic region.

Symptoms.—The early symptoms vary, depending on the etiology. In traumatic cases, initial pain, tenderness, and vomiting, as well as other evidences of local inflammation may be present. In other cases the early symptoms are more mild and not to be differentiated from the symptoms of chronic pancreatitis. In most cases the presence of a cyst is not suspected until it begins to cause enlargement of the abdomen. Clinically the cyst appears as a spheroidal, smooth, rather resistant, usually immobile mass, presenting in the hypochondriac region between the stomach and the colon, though, as already mentioned, it may present below the colon—hence below the umbilicus. Other symptoms result from the absence of the pancreatic juice from the intestine (azotorrhœa, steatorrhœa), as well as glycosuria. Certain symptoms result from pressure of the cyst or adhesions, such as neuralgic pains from pressure on the regional nerves, jaundice from obstruction of the common bile duct, difficulty in emptying the stomach and consequent dilatation of the stomach from obstruction of the pylorus or the duodenum, ascites and intestinal hemorrhage from pressure on the portal or the mesenteric vein, œdema of the lower extremities from obstruction of the vena cava, hydronephrosis from obstruction of the ureter, etc. Rupture of the cyst into the peritoneal cavity, the stomach, or the intestine may result in (sometimes) sudden decrease in the size of the abdomen, and watery discharges from the bowel.

Treatment.—The treatment consists of operation—drainage or extirpation of the cyst.

TUMORS OF THE PANCREAS.

Of the tumors of the pancreas, in themselves comparatively rare, carcinoma is the most frequent, though sarcoma and adenoma have been observed. Carcinoma of the pancreas is more common in men than in women, and occurs most frequently from the fortieth to the sixtieth year, but it has been observed in infancy and in advanced life. It is most common in the head of the pancreas, but it may be widespread or limited to the body or the tail. It may be small or very large, and frequently extends to adjacent structures by contiguity of structure, and gives metastasis to the regional lymph nodes and the liver. The growth is commonly an adenocarcinoma, usually of the so-called scirrhus variety.

Symptoms.—The early symptoms do not differ essentially from those of chronic pancreatitis. Epigastric pain is present in most cases, and is often paroxysmal. Jaundice, due to obstruction of the diverticulum of Vater or of the common bile duct, is a conspicuous symptom; it may develop gradually or suddenly, is intense and persistent, and is usually associated with enlargement of the gall-bladder (in contrast with an impacted calculus in the common bile duct, in which, in consequence of infection and consequent atrophy, the gall-bladder is usually small: Courvoisier's law). Other symptoms due to pressure on or adhesions to regional structures occur, but these are not different from those mentioned in connection with pancreatic cyst, although duodenal stenosis and consequent gastric dilatation (with nausea and vomiting) is probably the most common. Symptoms due to absence of the pancreatic juice are similar to those mentioned under chronic pancreatitis. In a small proportion of cases a tumor is palpable, and sooner or later the well-known anemia, emaciation, and cachexia of carcinoma intervene.

Diagnosis.—In the early stages, and, indeed, in the late stages in many cases, the disease is not susceptible of diagnosis. The most suggestive symptoms are pain, a palpable tumor, jaundice (with enlarged gall-bladder), anemia, emaciation, and cachexia. Carcinoma of adjacent viscera (pylorus, liver, colon, etc.) must be considered in the differential diagnosis.

Treatment.—The treatment is purely symptomatic. Cholecystenterostomy and even removal of the carcinoma have been practised in a few cases with good temporary results.

PANCREATIC CALCULI.

Etiology.—Pancreatic calculi are rare, scarcely more than one hundred cases having been reported. It occurs especially between the ages of thirty-five and forty-five years, and is apparently much more common in men than in women (26 cases in men, and 6 cases in women, in Oser's collection). As a rule, the calculi are small, often so small as scarcely to be discernible, but very large calculi (up to 2 to 4 cm. in diameter) have been observed. Usually there is more than one calculus, sometimes very many (upward of a hundred or more). They may be round, oblong, or irregular in shape, and smooth or rough. Usually they are composed of calcium carbonate, either alone or in combination with calcium phosphate. They are sometimes found in the pancreatic ducts at necropsy (a personal observation of my own); sometimes they become impacted in the duct of Wirsung, or in the common bile duct, or in the diverticulum of Vater. Thus they cause: (1) Chronic interstitial pancreatitis with marked overgrowth of the fibroid connective tissue and atrophy of the glandular tissue, and sometimes lead to dilatation of the ducts and large cyst-like formations, which rarely establish fistulous communications with adjacent organs, such as the stomach, the intestine, etc. (2) In consequence of secondary infec-

tion, they sometimes lead to abscess of the pancreas. (3) Sometimes, analogous to a similar process in the gall-bladder, they are said to result in carcinoma of the pancreas. The exact cause of the calculus formation is not known, although there is some evidence that bacterial infection of the mucous membrane results in their formation—analogous to the process in the gall-bladder. Indeed, calculi in the biliary passages and in the pancreas are sometimes associated.

Symptoms.—Pancreatic calculi often occasion few, if any, symptoms, especially when they lie dormant. Like gallstones, they produce symptoms when they move on from their place of birth (pancreatic colic), and when they produce secondary results in and about the pancreas.

Pancreatic colic is characterized by sudden severe pain in the epigastrium, radiating to the left mammary region, the left margin of the ribs, and the left shoulder, vomiting, and sometimes collapse. In some cases the pain is more diffuse, radiates to both sides, and is not to be differentiated from some attacks of gallstone colic. Following the attack, pancreatic calculi have occasionally been found in the stools. Confirmatory symptoms consist of salivation, hunger and thirst (said to come on after the colic), fatty diarrhœa, glycosuria, and other evidences of interference with the functional activity of the pancreatic juice. As a matter of fact, aside from pancreatic colic, the presence of pancreatic calculi is altogether insusceptible of diagnosis, the symptoms being those of its results—chronic pancreatitis, pancreatic abscess, etc. In the event of marked dilatation of the ducts a tumor may be palpable.

Diagnosis.—The diagnosis is suggested by epigastric pain (colic) often recurring and of obscure causation, and associated with glycosuria, and many muscle fibers and marked increase of neutral fat in the stools, and the absence of evidence of a newgrowth. The calculi are sometimes susceptible of recognition by the *x*-rays.

Treatment.—The treatment of the colic does not differ from that of gallstone colic—hot application to the abdomen, morphine and atropine hypodermicly, and partial or complete anesthesia (ether or chloroform) if necessary. Impaction of the stone with dilatation of the ducts or abscess formation calls for surgical intervention. Otherwise, the treatment is that of chronic pancreatitis, which also is largely operative.

DISEASES OF THE PERITONEUM.

ASCITES.

(*Hydroperitoneum.*)

Etiology.—Ascites, the accumulation of serous fluid in the peritoneum, may be due to general or local causes. The general causes comprise: (1) Those that determine dropsy in other parts of the body, such as chronic cardiac disease, sometimes antedated by chronic pulmonary

disease (such as emphysema), or pleural or mediastinal lesions; and (2) anemic and cachectic states, such as high-grade anemia, leukemia, chronic nephritis, amyloidosis, etc. The local causes comprise: (1) Portal obstruction, such as may occur in cirrhosis of the liver, advanced passive congestion of the liver, pylethrombosis, or compression or kinking of the portal vein; (2) chronic inflammation of the portal vein, tuberculosis or so-called simple; (3) abdominal tumors, such as carcinoma of the peritoneum, ovarian tumors, the enlarged spleen of leukemia, etc.

Symptoms.—The chief manifestation consists of a uniform or symmetrical enlargement of the abdomen. Secondary symptoms, due largely to embarrassed cardiac and pulmonary action from compression, are usually also present.

Examination reveals the abdomen to be more or less enlarged. In the recumbent posture, there is bulging of the flanks; the umbilicus may protrude; the superficial veins are often dilated and tortuous; the anastomosis between the epigastric and the mammary veins may carry the blood upward; in cirrhosis, a plexus of veins may centre in the umbilicus (caput medusæ). Percussion reveals dulness in both flanks and low down in the hypogastric and the inguinal regions, and tympany above —around the umbilicus; both the dulness and the tympany shift with change in the posture of the patient. Palpation reveals a distinct sense of fluctuation (which must not be confounded with a somewhat similar sensation transmitted by a fat and relaxed abdomen).

Diagnosis.—The diagnosis, as a rule, is readily made. One must exclude ovarian cyst, pancreatic cyst, a distended urinary bladder, and fluid feces in a dilated colon.

The fluid is usually pale amber in color, clear, somewhat opalescent, and alkaline. That which occurs in anemic and cachectic states has a specific gravity of 1010 or less, and contains less than 1 per cent. of albumin; that of obstruction (liver cirrhosis, etc.) has a specific gravity of 1010 to 1015, and contains from 1 to 3 per cent. of albumin; and that of inflammatory processes (exudates) has a specific gravity of 1015 to 1020 or more, and contains from 3 to 5 per cent. or more of albumin. Occasionally the fluid contains traces of urea, urobilin, purin bodies, sugar, etc. Inflammatory fluid is likely also to be turbid. Bacteriological studies show the fluid to be sterile, in the absence of infectious processes. Cytological studies reveal the cellular constituents of ordinary ascites (transudates) to be few and to consist of lymphocytes and endothelial cells, with a few erythrocytes and leukocytes; inflammatory fluids may reveal more polynuclear neutrophilic leukocytes. Cryoscopy reveals the freezing point to vary with the amount of albumin.

Occasionally the fluid is chylous, in consequence of obstruction and rupture of the lymphatics (it contains considerable albumin, less than 0.9 per cent. of fat, and perhaps some sugar); or it may be chyliform (no sugar, but considerable fat—5 per cent. or more); or it may be hemorrhagic (traumatism, rupture of minute vessels, petechial hemorrhages in the serous membrane, some forms of tuberculosis, carcinoma, etc.).

The percentage of albumin may be calculated by Reuss' formula—the percentage of albumin equals three-eighths of the last two figures of the specific gravity, minus 2.8. Rivalta's reaction may also be resorted to to differentiate transudates from exudates: to 50 c.c. of distilled water one drop of 50 per cent. acetic acid is added, and then one drop of the ascitic fluid; in the event of the fluid being inflammatory, on coming in contact with the acidulated water, it assumes the form of a bluish-white opalescent ring, which, as it broadens out, may become striated by many bluish-white lines.

ACUTE PERITONITIS.

Etiology.—Acute peritonitis may perhaps occur as a primary disorder, but it is very rare. Usually the condition is secondary—to disease of any of the abdominal organs; less commonly to diseases of more distant organs (by metastasis). The most important antecedent conditions are appendicitis, salpingitis, gastric and duodenal ulcer, cholecystitis, and pancreatitis; but any inflammatory, suppurative, or ulcerative lesion of the gastro-intestinal tract, the female genitalia, the liver, spleen, urinary organs, etc., may be followed by peritonitis. The important micro-organismal causes are: Bacillus coli, streptococci, staphylococci, Bacillus pyocyaneus, Diplococcus pneumoniæ, Bacillus proteus, Micrococcus gonorrhœæ, Bacillus typhosus, etc.; mixed infections are common.

Pathology.—The lesions may be circumscribed or diffuse; and fibrinous, serofibrinous, purulent, or hemorrhagic in character; rarely also putrid; or there may be only a small amount of thin, serous fluid; or the peritoneum may be only opaque and lustreless, no exudate having formed. The lesions may extend to the peritoneum from continuity of tissue, or from perforation into the peritoneum of a hollow viscus or abscess of a solid organ. Special interest attaches to localized peri-appendicular peritonitis, to pelvic peritonitis (the consequence of antecedent infection of the uterus or Fallopian tubes), to peritonitis about the pylorus, duodenum, and gall-bladder, and to subphrenic peritonitis. From these local lesions the peritonitis may become diffuse; but it may become diffuse also with such antecedent localizations as, for instance, after perforation in typhoid fever.

Symptoms.—In the localized forms of peritonitis the symptoms are largely those of the antecedent disorder, many of the manifestations of which are due to the associated peritonitis. The most important comprise abdominal pain, tenderness, and rigidity of the overlying muscles. Nausea, vomiting, and constipation are often present; and there is more or less fever, a rapid pulse (sometimes the hard, wiry pulse of the older writers), and leukocytosis; and a mass may be palpable. In the perforative cases sudden severe abdominal pain and collapse are usually observed. In the diffuse cases the pain and tenderness become more widespread, the abdomen distended and tympanitic, and free fluid may be more or less readily detected (physical signs of ascites). The pulse

becomes very rapid and weak, and the fever moderate or high (rarely only slight fever); nausea and vomiting may persist, and constipation is usually obstinate. The face becomes pinched, the eyes sunken, and the expression anxious (Hippocratic facies); the skin takes on a pallid, cyanotic hue, and may be bathed in continuous or frequently repeated sweats; the patient usually becomes delirious, and soon dies.

Diagnosis.—The diagnosis is usually quite apparent from the aforementioned symptoms. Special attention should be directed to ascertaining the antecedent condition answerable for the peritonitis. In addition to these disorders, in some cases it may be necessary, for a time at least, to consider the different types of colic (intestinal, renal, and gall-stone), intestinal obstruction, ruptured tubal pregnancy, embolism or thrombosis of the superior mesenteric artery, etc. *Subphrenic abscess* usually follows gastric (or duodenal) ulcer or carcinoma, or other suppurative or ulcerative lesions of the intestine; it is characterized by a collection of pus, or of pus and gas (subphrenic pyopneumothorax) between the liver and the diaphragm; and it is suggested by severe pain, nausea, vomiting, embarrassed respiration, chills, fever, sweats, and leukocytosis, and the physical signs of fluid or of fluid and air above the liver.

Prognosis.—In diffuse peritonitis death usually ensues within forty-eight hours. In the circumscribed cases the outlook varies with the extent and severity of the lesions and the treatment adopted.

Treatment.—The treatment, although modified somewhat by the antecedent disorder, is virtually that described for appendicitis. In the majority of cases the treatment is essentially and solely surgical.

CHRONIC PERITONITIS.

Etiology.—Chronic peritonitis is perhaps rarely a primary process, in which event it is usually set up by the tubercle bacillus. Commonly the condition is secondary to disease of any of the abdominal viscera, or it may be part of a multiple serositis.

Pathology.—Like acute peritonitis, the lesions may be circumscribed or diffuse. The circumscribed lesions often represent the residue of postacute localized peritonitis, and consist of more or less thick or thin fibrous adhesions or bands. These are especially common about the appendix, the female genitalia, the gall-bladder, pylorus, and duodenum, the liver, and the spleen; occasionally they involve the intestine, the mesentery, etc. They bind the organs involved to adjacent organs or tissues, and more or less seriously interfere with their functional activity. The diffuse lesions may consist of: (*a*) Widespread thickening and adhesions, which, uniting the intestines to the abdominal parietes and viscera, may lead to complete obliteration of the peritoneum (obliterative or deforming peritonitis). (*b*) Serous effusion, which may be slight, moderate, or great in amount, and is likely to be tuberculous in nature (p. 103). (*c*) Hemorrhagic effusion, which is most common in tuberculosis or carcinoma of the peritoneum.

Symptoms.—The circumscribed adhesions are responsible for much of the so-called chronic "indigestion," especially the adhesions about the appendix, the gall-bladder, the pylorus, and the duodenum. Pain may be dull and more or less persistent, or colicky. Eventually more or less general impairment of health and invalidism may ensue. A coil of intestine may become caught beneath a fibrous band and intestinal obstruction and strangulation ensue.

The diffuse cases with much fluid exudation are characterized by the physical signs of ascites; and, since most of the cases are tuberculous, there is usually also some fever, local pain and tenderness, and perhaps also muscular rigidity, emaciation, etc. A tuberculous focus elsewhere in the body may be detectable. Chronic obliterative or deforming peritonitis is suggested by palpable tumor-like masses and sacculated fluid, but it is rarely recognized during life.

Diagnosis.—The "dyspeptic" cases should be recognized by bearing in mind their etiological factors and associated conditions. Ascites and fever are significant of the more diffuse lesions (usually tuberculous).

Prognosis.—Some of the ascitic cases in young subjects tend to undergo spontaneous recovery, but in general the outlook in all cases is not reassuring.

Treatment.—The treatment of the tuberculous cases is mentioned on page 105; otherwise the treatment is surgical.

MULTIPLE SEROSITIS.

(Multiple Progressive Hyaloserositis, Nicholls; Polyorrhomenitis; Polyserositis; Indurative Mediastinopericarditis; Chronic Hyperplastic, Hyaline, or Deforming Peritonitis; Zuckergussleber; Zuckergussmilz; Zuckergussherz; Zuckergusslunge; Pericarditic Pseudocirrhosis of the Liver, Peck.)

Etiology.—The cause of multiple serositis is not well understood. Certain of the infective diseases, such as typhoid fever, pertussis, malaria, syphilis, and rheumatism, are set down as etiological factors in a number of the reported cases. In some cases the lesions are of an extremely obscure and unknown etiology, whereas in other cases the provoking agent is certainly the tubercle bacillus; other cases occur during the course of chronic nephritis.

Pathology.—Anatomically the distinguishing feature of these cases is a chronic fibrous (usually hyperplastic) inflammation of the several serous membranes of the body—a multiple serositis. In some cases the inflammation may remain localized to one or two serous membranes, but there is a marked tendency for all the serous membranes to become involved. The several serous membranes may become involved equally or unequally; usually the lesions are most marked in the pericardium and on both sides of the diaphragm. In some cases extensive calcification of the pericardium occurs. In cases in which there is but partial obliteration of the pleural or the pericardial sacs there is usually also

some fluid present. In many cases, however, not only is the pericardium thickened and obliterated, but it has also formed adhesions with the chest wall and the structures of the mediastinum, one or both pleuræ are thickened and obliterated, and the peritoneum is more or less thickened and adherent and contains a large quantity of fluid. In many cases the liver is adherent not only to the under surface of the diaphragm, but also to the stomach, the colon, the omentum, the anterior abdominal wall, etc. The lesions involve also the spleen, especially in those cases in which there are extensive adhesions in the left pleura. A distinguishing feature of the lesions is the development of thick, fibrous, almost cartilaginous, masses of connective tissue, that encase, compress, and often distort the organs, and give rise to an appearance suggesting confectioner's icing—whence the designation "iced liver," or sugar-iced liver (Zuckergussleber), etc. In many cases of primary pericarditis the liver is enlarged, smooth, and has sharp edges; later, with the development of the perihepatitis, the liver lessens in size, its surface becomes somewhat irregular, its edges rounded, and sometimes the entire organ becomes much distorted; still later, or in some cases earlier, the appearances characterized as nutmeg, with or without atrophic alterations, may supervene. Although some slight connective-tissue proliferation and some extension of newly formed connective tissue from the thickened capsule sometimes occur, the common absence of marked connective-tissue proliferation in the liver is noteworthy. In some cases the lesions of cirrhosis are encountered.

Symptoms.—Although the disease is characterized by slow, insidious, and intermittent course, the perihepatitis and the peritonitis dominate the clinical picture of the well-developed disease. Aside from this, that is, aside from the ascites, the clinical picture varies, depending upon the point of origin of the disease. In some cases the disease is ushered in with acute pericarditis, pleuritis (which may necessitate repeated tappings), or perihepatitis (circumscribed peritonitis), which subsides and leaves the patient apparently well for a number of years. Usually, however, the lesions of the pericardium and of the pleuræ are latent, and the first sensible evidence of the disease is ascites. In some cases of primary pericarditis slight and transient œdema of the legs may be present early, but this is not observed in many cases, inasmuch as the pericarditis is usually latent. When œdema does occur early, it usually soon subsides and does not recur until the ascites has been present for a long time, or until shortly before death; except shortly before death, it is inconspicuous when contrasted with the excessive ascites. Associated with early œdema, slight swelling of the spleen and disturbance of the gastro-intestinal tract may develop, but these usually subside with the ascites, and the disease, as a rule, is characterized by the entire absence of such symptoms until shortly before death. The ascites is characterized by the fact that it is excessive, that it necessitates repeated tappings, that it recurs rapidly after tapping, and that it may remain stationary (not necessitating tapping) for many years. After repeated tappings it

may be impossible to draw off much fluid again on account of the development of many adhesions.

In a few cases lesions of the peritoneum appear to be entirely absent, whereas, in other cases they are so slight as to appear inadequate to cause the ascites. These may be likened to similar cases of valvular disease of the heart, in which disproportionate ascites is the striking clinical feature, and in which the ascites is doubtless due directly to the congestion of the liver and its consequences, as maintained by Pick. In some cases both factors—the congestion of the liver and the peritonitis—are operative. In those in which the congestion is the major factor we should expect more or less swelling of the spleen and disturbances of the gastro-intestinal tract; in those in which the serositis is the major factor, we should expect an absence of such symptoms. Some distinction—anatomically, at least—may be drawn between cases in which the lesions are confined to the peritoneum and cases in which the lesions are more widespread. The cases in which the pericardium is unaffected reveal no congestive alterations in the liver; clinically, however, the two classes of cases are very much alike, and the "Zuckergussleber" may occur in both.

Diagnosis.—Recurring ascites in a person in otherwise good health, together with the disposition and ability to be up and about between tappings, should suggest the diagnosis. Of especial diagnostic significance are: a history of a previous attack of pericarditis, pleuritis, or perihepatitis; the early occurrence and subsequent disappearance of œdema of the legs; marked ascites with little or no œdema of the legs; an enlarged liver early (this may not occur), and a small and distorted but otherwise smooth liver late in the course of the disease; the absence or very late occurrence of marked enlargement of the spleen; a tendency to the repeated occurrence of attacks of pain, tenderness, rigidity, and possibly palpable and audible friction in the right hypochondriac region—attributable to attacks of perihepatitis; and the rapid recurrence of ascites after tapping. One should determine whether the lesions are confined to the region about the liver or are more widespread, involving the pericardium and the pleura. In these cases one must endeavor to dissociate the symptoms due to the serositis from those due to failure of the circulation induced by the pericardial part of the serositis and its consequences. In most of these cases prime importance attaches to the recognition of an adherent pericardium (page 485).

From cirrhosis of the liver the condition may be distinguished by the signs of adherent pericardium; the absence of the etiological factors of cirrhosis of the liver; the slow, insidious, protracted, and intermittent course; the long periods during which the ascites may remain stationary and the patient in good condition; the entire absence or transient presence of slight jaundice; the absence, in most cases, of portal congestion and gastro-intestinal disturbances; in some cases, the association of an enlarged, smooth, and firm liver, with marked ascites; and the fact that in many cases the patient survives a large number of tappings.

Prognosis.—The prognosis as to eventual recovery is bad, but the patient may live for many years.

Treatment.—The treatment is almost wholly symptomatic. Diuretics may effect the discharge of the too abundant fluid or a similar object may be achieved by free catharsis. Potassium iodide and mercury may be tried when there is a trustworthy history of syphilis, as well as in doubtful cases, since they hold out some hope of benefit. Tapping must be performed when necessary; usually it has to be done frequently, although occasionally the fluid remains at a standstill for long periods of time.

SECTION IX.

DISEASES OF THE URINARY SYSTEM.

Pathological Physiology.—The kidneys, together with the intestines, lungs, liver, and perhaps the skin, are charged with the function of excretion; that is, of removing from the body waste products of no further use in the economy. Perhaps the kidneys produce also a secretion, that is, a substance of service in the processes of metabolism; but this has not yet been definitely determined. The substances excreted by the healthy kidneys are especially the waste products of nitrogenous metabolism, certain mineral salts, pigments, and water. All of these are normally present in the blood, from which they are removed by the healthy kidney—the water and the mineral salts probably by the glomeruli, and the waste products of nitrogenous metabolism probably by the epithelium of the convoluted tubules. In disease the functional activity of the kidneys fails or becomes perverted, so that on the one hand, substances normally eliminated are retained and accumulate in the blood, whereas, on the other hand, the kidney becomes pervious to substances that ordinarily do not pass it. This failure of excretion is in some way related to the predominating symptoms of nephritis—oliguria, uremia (in part, perhaps, retention of waste products of nitrogenous metabolism), and œdema (in part, retention of sodium chloride); and the abnormal permeability of the renal epithelium is answerable for the other significant manifestation of nephritis—albuminuria. The albumin discharged with the urine in disease is the albumin of the blood, serum albumin and globulin; this fact (and others) shows a selective action on the part of the cells of the glomeruli, which in health never excrete serum albumin, although they may excrete a foreign protein, such as egg-albumen, peptone, or the Bence-Jones albumose (myelopathic albumosuria), etc. Furthermore, although when diseased the kidney usually suffers as a whole, certain parts seem to be unduly susceptible to the action of certain poisons, as the glomeruli to the toxin of scarlatina—which provokes a so-called glomerulonephritis, especially characterized by oliguria.

The urine, the totality of the excretory activity of the kidneys, is a measure not only of the functional capacity of the kidneys themselves, but also of other organs more or less remote from, though variously related to, the kidneys, and of the general metabolism. In diseased

conditions, then, the urine may exhibit (1) variations in its normal constituents, or (2) substances foreign to it in health (quantitatively or qualitatively); and these latter may be significant of (*a*) disease of the kidney and the urinary tract, or (*b*) disorders of general metabolism or of organs other than the kidney.

Variations in the Normal Constituents of the Urine.—The total daily volume of the urine is a measure of the local renal and the general blood pressure, of the amount of blood circulating through the kidneys, and of the activity of the renal epithelium and of the skin. The reciprocal relations existing between the kidneys and the skin are of much importance in determining the amount of water in the urine: other things being equal, the more the sensible and insensible perspiration (activity of the skin) the less the water in the urine, and *vice versa*. In health the renal epithelium responds to the amount of blood that circulates through the kidneys: an increased amount of blood results in an increased amount of urine, and the reverse. Thus, for instance, when in consequence of cold, the cutaneous vessels become constricted and the kidneys (and other abdominal organs) receive an increased supply of blood, as well as when, in consequence of vasomotor relaxation (nervousness, fright), more blood is determined to the kidneys, the urine is increased. In acute inflammation, however, the increased amount of blood is associated with swelling and other degenerative changes of the renal epithelium, and, the renal capsule being comparatively inelastic, compression of the renal capillaries, slowing of the circulation, and consequent oliguria result. In these circumstances, the effect of heat (as opposed to cold) in determining blood to the cutaneous surface and reducing renal blood pressure is resorted to and constitutes the basis of valuable therapeutic resources (flannel clothing, hot baths, warm climates, etc.). In these cases the administration of large amounts of water to flush the kidneys is neither wise nor efficacious, since before the kidneys can functionate properly the local circulatory conditions and the renal epithelium must become improved; for similar reasons, drugs that induce diuresis by increasing renal blood pressure are directly contra-indicated. The relief to tension is in part at least the rationale of decapsulation in subacute and chronic nephritis with oliguria. Oliguria results in all conditions of lowered renal blood pressure, as in chronic passive congestion, in shock, etc.; excretion ceases if the pressure is reduced to 40 mm. Hg. The polyuria of chronic indurative nephritis is due largely to increased general blood pressure—which is a manifestation of adaptation on the part of nature to compensate for changed conditions, and it should not, therefore, be too much reduced, since reduction may lead directly to oliguria and uremia. Polyuria also may be due to an effort on the part of the kidney to excrete a foreign substance such as sugar (diabetes), or to excrete the normal amount of normal ingredients when the kidneys become reduced in capacity (diabetes insipidus).

The acidity of the urine is due to acid phosphates and organic acids. Special clinical importance attaches to β-oxybutyric acid and its deriva-

tives in the acidosis of diabetes and other conditions. The nitrogenous constituents of the urine make up about 16 grams daily on an ordinary diet; about 85 per cent. is urea, 1 to 3 per cent. uric acid, 2 to 5 per cent. ammonia compounds, and the remainder purin bases, creatinin, hippuric acid, etc. Of special clinical importance is the fact that the nitrogenous excretion in health is proportionate to the nitrogenous intake; that it is futile to estimate the output of urea (formed largely in the liver from ammonia compounds) without at the same time estimating the total nitrogenous output and knowing the nitrogenous intake (the protein content of the food); and that although the ratio of uric acid to urea is usually about 1 to 70, it varies considerably in health, depending upon the diet and other factors. The ammonia output has come to be recognized as a valuable measure of the presence or absence of acidosis, and is of much clinical importance in the acid intoxication of diabetes, pregnancy, etc.

Chlorides, present chiefly as sodium chloride, are derived from the food, and in health the output is proportionate to the intake. In some cases of parenchymatous nephritis the renal epithelium appears to be incapable of excreting chlorides, which being retained constitute at least one factor in the causation of œdema. The phosphates also are derived largely from the food. The condition spoken of as phosphaturia is usually due not to excessive output, but to lessened acidity of the urine and precipitation of earthy phosphates; rarely there may be an excessive output (so-called phosphatic diabetes) in neurasthenia, tuberculosis, diabetes, leukemia, etc.; in some cases it seems to be associated with disturbances of the calcium metabolism.

The sulphates result from decomposition of proteins, and occur as inorganic sulphates, ethereal (organic) sulphates, and neutral (unoxidized) sulphates. The ethereal sulphates result in part from metabolic processes, and in part from the union of sulphuric acid and certain aromatic products of intestinal putrefaction (phenol, indoxyl, skatoxyl); to some extent, then, they are a measure of intestinal putrefaction, and with reservation may be thus interpreted clinically (indicanuria).

Oxalates (chiefly calcium) are derived largely from vegetable foods (spinach, rhubarb), but in part from metabolic processes. An increase, spoken of as oxaluria (sometimes spurious, due to precipitation in consequence of a poverty of magnesium salts), is sometimes associated with neurotic disorders, indigestion, etc.; and the salts sometimes constitute renal calculi. Urobilin is normally present in small amounts, and it is increased (urobilinuria) in cases of excessive erythrocyte destruction, (pernicious anemia, certain forms of hemolytic jaundice, etc.).

Abnormal Constituents in the Urine.—Under varying circumstances the urine contains a large number of substances foreign to it in health, quantitatively or qualitatively; of these, the most important are albumin, tube casts, blood, and pus—which are of much significance in the diagnosis of disease of the kidney and the urinary tract; furthermore, glycosuria and ketonuria (major phenomena of diabetes) also are common;

and there are other more or less important anomalies of urinary excretion.

Thus, the urine may be abnormally colored. Deep yellowish or orange-colored urine may be due to: an excess of urochrome (the normal urinary pigment); urobilin, due largely to the action of intestinal bacteria on bilirubin, and found in conditions of excessive hemolysis, pernicious anemia, hemolytic jaundice (sometimes inaptly called urobilin jaundice) constipation, etc.; uro-erythrin (liver disease, fevers, etc.); chrysophanic acid (rhubarb, senna); santonin, etc. Pinkish or reddish urine may be due to oxyhemoglobin (hemorrhage), uro erythrin (pinkish uratic sediment), eosin, rosanilin, etc. Greenish urine is due to the presence of bile pigment (biliverdin) or methylene blue; bluish urine is practically always due to methylene blue. Brown or black urine may be due to methemaglobin (hemorrhage—which causes smoky urine; paroxysmal hemoglobinuria—so-called black-water fever); to an excess of bile pigment (especially biliverdin); to an excess of indican (blackness on standing; higher oxidation products of indol and not indoxyl sulphate, which is colorless); to melanin or melanogen (disseminated melanotic tumors); to the ingestion of carbolic acid, creosote, salicyl compounds, etc.; to hematoporphyrin, which causes a port-wine or blackish discoloration of the urine as one of a series of toxic phenomena that occur especially in women after the long-continued use of sulphonal or trional, and perhaps under other circumstances (lead poisoning, intestinal hemorrhage); and to alkapton (alkaptonuria, a brownish or blackish discoloration of the urine upon exposure to the air, associated with reduction of Fehling's solution, but no other "sugar" reaction, no fermentation, and no rotation of polarized light); and ochronosis (blackish pigmentation of the cartilages), which is a congenital vice of metabolism (incomplete combustion of aromatic derivatives of proteins), resulting in the presence in the urine of homogentisic acid; the linen may be stained black by the urine, and ultimately the cartilages of the ear, the sclerotics, and even the skin may become obviously pigmented.

Other rare urinary anomalies comprise chyluria, which may be parasitic (true chyle) or non-parasitic—opaque, whitish, opalescent, milky urine; lipuria (fat in the urine), which may occur after an excess of fatty food, or of cod-liver oil, in fat embolism after fractures, in fatty degeneration, phosphorus poisoning, prolonged suppuration, diabetes, chronic nephritis with fatty degeneration, pyonephrosis, spurious chyluria, etc.; choluria, the presence of bile pigments (bilirubin and biliverdin in cases of jaundice); cystinuria, of interest because rarely it may lead to the formation of cystin calculi; fibrinuria, the formation in the urine of a jelly-like mass, occasionally associated with marked albuminuria; pneumaturia (the presence of gas in the urine), which may result from trauma or the instrumental introduction of gas into the urinary tract, gas from the intestinal tract (vesico-enteric fistula), or fermentation of urine in the bladder by gas-forming organisms (Bacillus coli, Bacillus lactis aërogenes, Bacillus aërogenes capsulatus)—usually found in diabetic subjects.

Uremia.—Uremia is a form of auto-intoxication that occurs in nephritis and in certain conditions associated with anuria. The toxic agent is not known. Commonly the uremic manifestations are ascribed to the activity of retained poisonous metabolic products that should be and in health are eliminated by the kidneys, and these products are often assumed to be nitrogenous in nature. The poison is not urea, uric acid, potassium or sodium salts, creatinin, etc., since these may be injected into the body without evil consequences. The condition has been attributed also to an acid intoxication (lessened alkalinity of the blood), to urotoxins (Bouchard), to toxemia of the blood (Hughes and Carter), to œdema of the brain and meninges (Traube), to failure or perversion of a supposed internal secretion of the kidneys; to nephrolysins, etc. It is not improbable that it results from the products of perverted metabolism or from non-detoxification of metabolic products normally robbed of the poisonous products (by the liver, for instance). The cerebrospinal fluid is often under increased pressure.

Clinically uremia may be acute or chronic, or latent (in conditions of obstruction of the ureters and anuria). Acute uremia may occur in any type of nephritis, but it is especially common in acute and chronic diffuse non-indurative nephritis; it is often preceded by chronic manifestations (headache, restlessness, dyspnœa, insomnia, etc.); it develops suddenly, is likely to last only a short time, and commonly leads to death. Chronic uremia is most common in chronic diffuse indurative nephritis; it develops insidiously, is mild in its manifestations, is likely to last a long time, may disappear and recur, but is likely ultimately to lead to acute manifestations and death.

The major symptoms of acute uremia are nervous in nature and consist of (*a*) convulsions (not to be distinguished from those of epilepsy, except probably the absence of the initial cry); (*b*) coma, which, aside from that which attends and follows the convulsions, may come on insidiously and last several days or weeks, and lead usually to death, but sometimes eventuates in recovery; and (*c*) psychic derangements (melancholia, delusional insanity, mania, etc.). Hemiplegia or monoplegia sometimes occurs, and may be due solely to œdema of the brain and meninges. Other less serious and more chronic nervous manifestations comprise headache, hemicrania, vertigo, insomnia, tinnitus aurium, amaurosis, languor, neurasthenic phenomena, muscular pains and twitchings (especially in the calves), joint pains, etc. Dyspnœa (renal asthema) is often an early and obtrusive sign; it may be continuous or paroxysmal (developing especially at night), or of the Cheyne-Stokes type. Nausea and vomiting (sometimes uncontrollable) are common, and may be associated with diarrhœa; these may result from toxic irritation of the medullary centres or from gastro-intestinal irritation provoked by the excretion of urea into the stomach and its decomposition into ammonia. Stomatitis, a coated tongue, and foul breath are frequent. The temperature is usually subnormal, although a so-called uremic fever has been described. Uremia is usually easily recognized, but in the event of coma one must

eliminate alcoholism (obvious alcoholism, alcoholic habits, less deep coma), opium poisoning (contracted pupils, infrequent respirations, no convulsions), diabetic coma (ketonuria, perhaps glycosuria, fruity odor to the breath, Kussmaul's dyspnœa), and apoplexy (sudden onset of hemiplegia, flapping of one cheek, conjugate deviation of the eyes, perhaps unequal pupils). Apoplexy, however, is common in chronic nephritic subjects, so that albumin and casts in the urine are not conclusive evidence of the one or the other condition; nor do they exclude diabetic coma, since at least one-half of the diabetic subjects have also chronic nephritis.

Cardiovascular Changes in Nephritis. — The cardiovascular changes in nephritis are of extreme interest and importance; as a matter of fact, they dominate the clinical picture of the disease and they assume commanding position in the prognosis and in the treatment. The lesions are widespread, involving the heart, the large and the small arteries, the capillaries, sometimes even the veins. The lesions of the heart involve the muscle and find their expression in hypertrophy. With this, at times, there is associated some interstitial myocarditis, with increase of the intermuscular fibrous trabeculæ, especially in the neighborhood of many of the smaller coronary arteries, which frequently are obliterated. In some cases, especially late in the course of the disease, fatty degeneration of the heart muscle also may supervene. Valvular lesions are extremely uncommon. Those that are found in cases of renal disease are either an antecedent condition, or they result from mechanical causes, from dilatation of the heart, the lesion being one of relative insufficiency. The hypertrophy of the heart is predominantly a lesion of the left ventricle. In the majority of cases the left ventricle alone is affected; in the remainder of the cases both ventricles are affected. The right ventricle is never affected alone. Simple hypertrophy of the left ventricle is the first event; later, this is associated with some dilatation—eccentric hypertrophy. The longer the nephritis continues (the longer the patient lives), the more certain the development of consecutive enlargement of the right ventricle, the increased pressure in the left ventricle, coupled with degenerative alterations in the heart muscle, leading to increased pressure in the right ventricle. Here also the hypertrophy is of the eccentric variety. Predominating dilatation of either or both ventricles is an unusual event until toward the termination of life; its development is of ominous moment—it usually presages an early fatal termination. It has been stated by Senator and others that the hypertrophy of the primarily contracted kidney is of the simple variety, whereas that associated with the secondarily contracted kidney and with the arteriosclerotic kidney is of the eccentric variety—hypertrophy with dilatation. But this statement scarcely permits of general application.

This hypertrophy of the heart occurs in all forms of nephritis, but not in all cases. In none of the varieties, however, does it so dominate the clinical picture as it does in chronic indurative or interstitial nephritis. It may be expected from the time of the contraction or atrophy of the

kidney, be it of the primary or the secondary variety. Thus it occurs early in the primary form, but its occurrence may be (but usually is not) much delayed in the secondary form. It may, however, occur in chronic diffuse non-indurative (or parenchymatous) nephritis, and it is observed in some cases of acute nephritis, particularly in some of the cases of acute nephritis following the infections. In some of the chronic cases in which it should be expected, for some more or less inexplicable reason it may not occur, at all events not to a noteworthy degree. These, however, are the unusual cases. The absence of the hypertrophy may be due to faulty nutrition—the heart not being sufficiently well nourished to enable it to hypertrophy; to sclerosis of the coronary arteries; to the vicarious action of one of the other emunctories removing those excrementitious products ordinarily eliminated by the healthy kidneys, etc.

The changes in the arteries are those of augmented blood pressure, and arteriosclerosis and atheroma. These changes, though widespread, are not distributed uniformly throughout the body. In many cases they are most marked in the kidneys; in a few cases they appear to be almost confined to them. In many cases small miliary aneurysms develop, especially in the brain, and rupturing, frequently lead to the fatal termination.

The cause of these cardiovascular alterations has not been thoroughly worked out. Considerable discussion still obtains with reference to whether the primary alterations are in the arterioles (stopcock action of Johnson) or in the capillaries (the arteriocapillary fibrosis of Gull and Sutton). Doubtless, the conditions differ in different cases. Thus, arteriosclerosis itself may be the primary event (and this seems to be especially common in this country), and it leads to both the kidney alterations and the cardiac hypertrophy. In this instance the disease is a widespread process, and its etiology is the etiology of arteriosclerosis. However, in many cases the nephritis is the primary event, and leads to the cardiovascular alterations. Whether these are mechanical or chemical in nature is still a matter of discussion. Augmented blood pressure is undoubtedly at the bottom of the process, but the old view of Traube and of Cohnheim that this resulted from an effort on the part of the cardiovascular system to maintain a normal or adequate flow of blood through kidneys, the capillary distribution of which had become reduced by interstitial overgrowth, has been largely given up. It has been modified to some extent by certain observers, who point out that these cardiovascular lesions occur most commonly in association with disease of the glomeruli, and that the function of the glomeruli seems to be that of filtration dependent upon the blood pressure (as contrasted with the glandular secretory activity of the renal epithelium); and they suggest that when the glomeruli become more or less inactive in consequence of disease, the regulatory mechanism of the body attempts to compensate therefor (to promote glomerular elimination) by increased blood pressure—which becomes general. On the other hand, it has been suggested that the primary increase of blood pressure is chemical

in origin, is due to the angiotonic properties of the adrenal body; that the condition results from hyperadrenalism. Alterations, interpreted by some observers as hyperplastic in nature, have been found in the adrenals in nephritis (although it has not yet been determined whether they are the cause or the result), and a pressor substance has been found in the disordered kidneys. Whether this is adrenalin or a substance allied thereto, and whether this is the result of increased formation or of retention (reduced elimination by diseased kidneys), has not been determined. Perhaps certain enterogenic or other poisons not detoxified by the liver may be at the bottom of the process.

The Functional Capacity of the Kidneys.—The most obvious is at the same time the best test of the functional capacity of the kidneys—the daily amount of the urine; for suppression of urine is a certain sign of renal inadequacy (whether the primary cause be resident in the kidneys themselves or elsewhere, such as the cardiovascular system). In addition to oliguria or anuria, albuminuria, cylinduria, and uremia are important manifestations of functional incapacity of the kidneys; these are discussed under separate headings.

Other tests of the functional capacity of the kidneys comprise: (1) The indigo-carmine test. This consists of the intramuscular injection (gluteal muscles) of 20 c.c. of a 0.4 per cent. aqueous solution of indigo-carmine, and ascertaining, preferably by means of cystoscopy, the time of beginning excretion (light blue color to the urine) and the time of maximum excretion (dark blue color) of the indigo-carmine. Normally the excretion begins within twenty minutes (averaging perhaps fifteen); if the excretion is delayed beyond twenty minutes, certainly if beyond thirty minutes, serious or grave disease of the kidneys is almost certain. This test is especially significant of surgical diseases of the kidney or obstruction of the ureter. (2) The methylene blue test. This comprises the intramuscular injection of 1 c.c. of a 5 per cent. aqueous solution of methylene blue; the collection of the urine after the lapse of one-half hour, one hour, and hourly thereafter; and noting the time of the appeareance of a bluish tint to the urine, the time of maximum coloration, and the time of disappearance of the coloring. Normally a slight tint may be noticed in the first specimen, certainly at the end of one hour; the maximum coloration occurs at the end of three or four hours, and the urine is free of coloring at the end of thirty-six to forty-eight or sixty hours. Delay of beginning excretion beyond one hour and of maximum coloration beyond the fourth hour, and continuation of excretion, as may occur for five or six days, is indicative of deficient functional capacity. (3) The phloridzin (glycosuric) test. This consists of the intramuscular injection of 1 c.c. of a 0.5 per cent. aqueous solution of phloridzin, and the collection of the urine as in the methylene blue test. In normal subjects glycosuria may be detected at the end of one-half to one hour, and it disappears at the end of two to four hours. In disordered renal epithelium this glycosuria may be delayed or may not occur at all. (4) Cryoscopy, or the deter-

mining of the freezing point of the urine, is a test of its molecular concentration, particularly of its sodium chloride ions and protein constituents. Normally, the freezing point of the urine varies between —1.3° and —2.3° C.; in diseased states the freezing point is —1° or less than —1° C. (hyposthenuria), but since this depends upon various factors (sodium chloride, protein constituents, urates, etc.) it loses clinical value. Comparison of the freezing point of the urine with that of the blood, which normally is —0.56° C., is of more value. (5) The electrical conductivity of the urine. This varies with the contained ions, but is influenced by molecules that do not dissociate (sugar, etc), so than its clinical value is slight. All of these tests are of more value in determining the relative functional capacity of the two kidneys (separated urines) than the absolute functional capacity of both kidneys together, and they are especially applicable in surgical conditions of the kidneys and ureters.

ANURIA.

Anuria, or suppression of urine, in which no urine enters the bladder, should be distinguished from retention of urine, in which urine, though present in the bladder, is not or cannot be voided. In retention (unless as in typhoid fever, for instance, the sensibility is benumbed) there is likely to be complaint of distress referred to the bladder, although in some of the cases, such as elderly men with enlarged prostates, some overflow of urine may be associated with extraordinary retention of residual origin altogether unknown to the patient; in any event, however, examination of the bladder region and catheterization will reveal the presence or absence of urine in the bladder. The causes of retention are some mechanical obstruction (urethral stricture or stone, enlarged prostate, pregnant uterus, etc.) or some functional incapacity (paresis from overdistention, functional and organic diseases of the nervous system, etc.). Anuria may be obstructive or non-obstructive.

Obstructive Anuria.—Obstructive anuria results from compression of both ureters, as by a pelvic growth, such as a uterine carcinoma, for instance; blocking of both ureters by renal calculi, blood clots, tuberculous material, tumors, twists or stricture, etc.; or blocking of one ureter in cases in which the other kidney is absent or functionless. The anuria in these cases, which are sometimes spoken of as "latent uremia," may persist for some time (one or two weeks or, rarely, longer) and the patient suffer comparatively little discomfort. Uremia (convulsions, coma, or psychoses) and œdema are quite unusual. The treatment is surgical.

Non-obstructive Anuria.—Non-obstructive anuria occurs: (1) In acute toxic degenerative and diffuse nephritis, such as occurs in the infective diseases (scarlet fever, yellow fever, cholera, etc.) and in different poisonings (phosphorus, lead, cantharides, etc.); (2) in cardiovascular failure, and passive congestion; (3) following severe, especially abdom-

inal injuries and operations (sometimes even the passing of a catheter), particularly in persons past middle life whose kidneys and bloodvessels are somewhat damaged; and (4) as a manifestation of hysteria (although hysterical retention and deception are more common). Aside from hysterical anuria, in which, as in obstructive anuria, there is usually a remarkable absence of symptoms, non-obstructive anuria is of serious moment—being one of the manifestations of serious degeneration of the kidney or nephritis. The treatment is that appropriate in acute diffuse nephritis.

ALBUMINURIA.

Albuminuria is a term used to denote the presence in the urine of different protein substances—serum albumin, serum globulin, nucleo-albumin, albumose, peptone, fibrin, and hemoglobin. While formerly regarded as diagnostic of Bright's disease, albuminuria is now known to be dependent upon many different diseased conditions. Large amounts of albumin in the urine, however, practically always indicate serious kidney disease, but serious disease of the kidneys may exist for a long time with little or no albumin in the urine. In the majority of cases of albuminuria the albumin is moderate in amount, and one generally has to determine whether the albumin is due to disease of the kidneys, to disease of some other organ, or to both. The importance and significance of albuminuria are always enhanced by the associated presence of tube casts, but both albumin and casts may be present without serious disease of the kidneys, and serious forms of Bright's disease may exist with at least temporary absence of albumin and casts from the urine. The differential diagnosis of albuminuria, therefore, is of considerable importance. The following forms of albuminuria may be distinguished: (1) Extrarenal, spurious, accidental, or factitious albuminuria; (2) so-called functional, physiological, or cyclic albuminuria; (3) albuminuria without noteworthy alterations in the kidneys; and (4) albuminuria with noteworthy alterations in the kidneys.

Extrarenal, Spurious, Accidental, or Factitious Albuminuria.—In these cases the albuminuria is but a subsidiary factor and entirely dependent on the admixture with the urine of different albuminous fluids—blood, pus, chyle, or seminal discharges. Chyle is a very rare constituent of the urine, and when present will scarcely fail of recognition. Seminal discharges in the urine are of more common occurrence, and should not escape careful observation. The greatest interest attaches to cases of albuminuria associated with blood and pus in the urine—the result of inflammation and ulceration of, and of hemorrhage from, any part of the genito-urinary tract, especially the renal pelvis, ureters, bladder, urethra, vagina, uterus. In these cases the amount of albumin is small—rarely more than 1 part per 1000 (Esbach's method), and it is proportionate to the amount of blood and pus. A disproportionate amount of albumin should always excite a suspicion of the co-existence of nephritis.

The presence of tube casts in the urinary sediment is of the greatest importance—their persistence being clear evidence of nephritis, and indicating that at least a part of the albumin in the urine is due to circulatory and tissue changes in the kidneys. One must avoid error in presuming the non-existence of nephritis in cases in which a small amount of albumin and casts are associated with a few leukocytes or pus corpuscles. In most of these cases the albuminuria is due in great part to nephritis—a few leukocytes or pus corpuscles being regular constituents of the urine in Bright's disease. (For additional information, see hematuria, pyuria, and nephritis.)

So-called Functional, Physiological, Cyclic, Intermittent, Paroxysmal, Dietetic, Postural (Orthostatic), and Adolescent Albuminuria.—The occurrence of a "physiological" albuminuria is now generally conceded. It is found especially in young, more particularly male, subjects under twenty-five years of age. It is usually detected accidentally, as, for instance, at examinations for life insurance, or for other certificates of health. In some cases it appears to be constant, and it lasts for a relatively long time; in other cases it is intermittent—occurring at stated or indeterminate periods. In many of the cases the presence and the amount of the albumin are much influenced by certain circumstances—posture (whence it is sometimes absent on arising in the morning and present later in the day), exercise, emotional and other excitement, cold baths, certain articles of diet (especially proteins, but also large amounts of ordinary mixed foods), prolonged anxiety, college and university examinations, etc. In diagnosticating this form of albuminuria considerable circumspection must be exercised. The patient should be a young subject; the urine should be normal in amount (certainly not increased), normal in color, and normal in specific gravity (certainly not of lessened specific gravity); the amount of albumin must be small—less than 0.5 per 1000 (Esbach); the urine must contain no other abnormal constituent; there must be no local disease of the genito-urinary tract, such as gleet, spermatorrhea, leucorrhea, etc., that might give rise to spurious albuminuria; every bodily organ must be examined and found to be in perfect health—in particular there must be no hypertrophy of the left ventricle of the heart, no accentuation of the aortic second sound, and no increased arterial tension; nor may there be any evidence of disorder of the heart, lungs, or liver that might occasion congestion of the kidneys; there must have been no past, nor may there be any present, œdema, uremic manifestations, or retinal changes; and at the most there must not be more than an occasional hyaline cast. Even in cases that meet these requirements much caution must be observed, and the diagnosis must not be made until the patient has been long under observation, until both the morning and the evening urines have been repeatedly examined, and until the albumin is found to vary with the influences under which it develops. And when the diagnosis has been made one should stand willing to renounce it immediately on the development of signs of disease likely to be attended by albuminuria. Caution is

always necessary, inasmuch as albumin may be temporarily absent, even for long periods in certain cases and at certain stages of chronic indurative (or interstitial) nephritis, whereas, in other cases it may be present in such small amounts as to require delicate tests for its detection. More than a small amount of albumin always suggests a latent nephritis that for the time being may reveal itself by no sign other than the albuminuria. The caution necessary in the diagnosis of these cases is evident from the statement of Senator, who believes that most cases of so-called cyclic or functional albuminuria are really cases of latent nephritis, and that no case of albuminuria may be regarded as functional or physiological if the albumin exceeds 0.4 or 0.5 part in 1000. The diagnosis of functional albuminuria in late adult life had best be dispensed with entirely, as practically without exception small amounts of albumin in the urine of such subjects means chronic nephritis (excluding cases of extrarenal albuminuria).

The cause of this functional or cyclic albuminuria is not well understood. It has been attributed to vascular alterations (congestion) of the kidneys, and to disturbances in metabolism, in consequence of which albumoses, excessive in quantity or abnormal in quality, are formed. That many of these cases are really cases of physiological albuminuria may well be doubted. Some of them are certainly cases of spurious albuminuria. In most cases it is likely that the glomerular epithelium suffers some nutritional disturbance. In some cases the renal epithelium is probably unduly sensitive. These cases may be confounded with the rather unusual cases in which following an acute nephritis in early childhood a trace of albumin persists throughout life, but the patient lives to a good old age without other manifestations of nephritis developing. Such persistent albuminuria, of course, indicates slight alterations in the renal epithelium, and in case careful injuiry is not made into the antecedent history of the patient the condition may be regarded as "physiological" albuminuria.

Albuminuria without Noteworthy Alterations in the Kidneys.—A slight amount of albumin may be found in the urine in a great variety of morbid conditions without noteworthy alterations in the kidneys. Thus it may be found in different blood conditions, such as the anemias, leukemia, purpura, scurvy, etc.; in certain forms of intoxication, such as lead poisoning, mercurial poisoning, etc.; in conditions associated with the presence of substances, such as sugar and bile-pigment in the blood; after the administration of anesthetics; in fever conditions, and in certain nervous disorders—epilepsy, apoplexy, meningitis, tetanus, exophthalmic goitre, injuries to the head, etc. In most of these cases the albuminuria is attributable to nutritional disturbances in the glomeruli and renal epithelium induced by defective blood supply; in some cases to the result of nervous influences. The true nature of these cases is usually readily recognized. The albuminuria is slight and temporary, as a rule; the primary disorder is sufficiently obvious, and there is no other manifestation of Bright's disease. It is important, however, to point out that the

conditions in the kidneys found in some of the intoxications and in fever states (toxic degeneration of the renal epithelium) merge gradually into the well-defined lesions of nephritis—that there is no sharp dividing line between them.

Albuminuria with Noteworthy Alterations in the Kidneys.—In this group are included the cases of active and passive congestion of the kidneys, cases of toxic degeneration of the kidneys (most of the cases of febrile albuminuria), the kidney of pregnancy, amyloid degeneration of the kidneys, the acute and chronic suppurative and non-suppurative inflammations of the kidneys, and tumors of the kidneys. In all of these cases the albuminuria is due to some more or less severe, temporary or permanent, damage to the kidneys, and the albuminuria acquires its clinical significance and diagnostic importance from the attendant phenomena. In acute congestion of the kidneys the albumin is small in amount, the urine is decreased in quantity and frequently more or less hemorrhagic, and the causative factor usually can be determined. In most cases, however, acute congestion of the kidneys represents but the early stage of acute nephritis, from which it is scarcely to be differentiated. In chronic congestion the albumin varies in amount in different cases. In chronic heart and lung diseases it is usually small in amount, but in cases of chronic congestion due to obstruction of the renal veins by a tumor or ascites, etc., the amount may be large. The diagnosis of chronic congestion is to be based on the attendant phenomena and not on the amount of albumin. Toxic or febrile albuminuria is an interesting condition that merits much attention. The albumin in these cases is due to the direct action of some toxin on the renal epithelium rather than to the fever, though this may be a contributing factor; in the one case the toxin induces marked degeneration of the renal epithelium, in another case it leads to severer lesions—to an acute nephritis. In toxic albuminuria the albumin is slight or moderate in amount and transitory, disappearing with the intoxication or infection that gives rise to it.

ALBUMOSURIA.

True albumose is sometimes found in the urine—during postpartum involution of the uterus (physiological), during fevers, during absorption of pneumonic exudates, in phosphorus poisoning, in acute yellow atrophy of the liver, and in other conditions of autolysis—to which it is attributable. True peptonuria has occasionally been found in pneumonia, tuberculosis, etc. Globulin is occasionally found in association with large amounts of albumin; it may occur alone.

The Bence-Jones protein (myelopathic albumosuria; Kahler's disease) is a protein foreign to the blood in health; it is formed in multiple myeloma, of which it is diagnostic; and it may be excreted by normal kidneys without impairment of their epithelium (selective action). It is distinguishable from ordinary albuminuria by the production of cloudiness in the urine when heated to 50° C., by a flocculent precipitate

between 50° and 60° C., by solution of the precipitate at higher temperatures (especially upon the addition of acetic acid), and by reprecipitation upon cooling.

CYLINDRURIA.

The term cylindruria is commonly employed to designate casts of the uriniferous tubules (cylinders, tube casts), although perhaps other casts might be included within the scope of its meaning; these, however are casts of the renal pelvis, ureter, urethra, etc., and since they are usually made up of blood, they are referred to under hematuria. Tube casts basically are made up of coagulated serum or a serum-like substance exuded by the renal epithelium when irritated, or perhaps they result from the interaction of serum and another substance elaborated or liberated by disordered renal epithelium. The basic (hyaline) cast may become altered or attach various sorts of cells and debris, so that different kinds of casts may be distinguished—hyaline, waxy, granular (pale and dark), and cellular; of the cellular casts, sometimes (but by no means always) the type of cell may be distinguished—epithelial, leukocytic, erythrocytic; an obvious fatty degeneration of the cells, as well as free fat droplets, suggests the term fatty cast; and some casts apparently consist wholly of leukocytes or erythrocytes without the basic hyaline material.

Considerable diagnostic significance attaches to the presence of casts. They suggest mere irritation (hyaline cast) or more complete destruction and desquamation of the renal cells (epithelial, granular, and fatty casts). A few hyaline casts may occur in conditions of slight nutritive disturbance, such as occurs in congestion; and since the renal epithelium is not markedly disturbed in chronic interstitial nephritis, few casts are encountered. Many, especially epithelial and granular, casts indicate marked alteration and desquamation of the renal epithelium, such as occur in parenchymatous nephritis. Casts and albumin may and usually do occur together, but either may occur alone; casts alone are the more significant of changes in the renal epithelium. Few casts for a short time signify a mere transitory disturbance; the continued presence of even a few casts means chronic nephritis; many cellular (epithelial) casts mean serious parenchymatous involvement of the renal epithelium; erythrocytic casts, an acute exacerbation; and a lessening number of cellular and erythrocytic casts, a subsiding process, etc.

Too much importance must not be attached to a few hyaline or cellular casts in a single specimen of urine; they are common in transitory conditions, such as anemia, various intoxications, certain infections, etc., and although they indicate some disturbance in the renal epithelium, they subside with the initiating cause: the renal condition is not nephritis, as is too often supposed, and the kidney often becomes entirely normal again. Casts should not be mistaken for pseudocasts or cylindroids—long hyaline or longitudinally striated objects with undulating outline and tapering ends; these are mucoid in nature and not related to true casts.

HEMATURIA.

Blood in the urine may come from any part of the genito-urinary tract. Care must be exercised to exclude menstrual blood and urine stained with logwood, rhubarb, etc. Small amounts of blood in the urine may escape naked-eye observation; moderate or large amounts impart a dark brownish or smoky color; while very large amounts may appear as bright or dark red blood. The blood may be recognized by microscopic, spectroscopic, or chemical examination.

Etiology.—Hematuria may be due to: (1) Disease of the urethra—usually traumatic (catheterization, bougieing, passing of a calculus, etc.), of which there is a history. The blood is pure and unmixed, and escaping at any time may stain the patient's linen. (2) Disease of the prostate—benign or malignant enlargement, of which hematuria is sometimes one of the earliest manifestations. (3) Disease of the bladder—calculus, tumor (benign or malignant), ulceration (simple or tuberculous), parasites, foreign bodies, varicose veins, etc. Blood from the bladder is usually quite apparent, not intimately mixed with the urine, often irregularly clotted, and may escape at the end of micturition; bladder epithelium and blood corpuscles are detected by microscopic examination (hemoglobinuria does not occur alone); irrigating the bladder results in securing a blood-tinged fluid; and the local symptoms are referred to the region of the bladder. (4) Disease of the kidneys—chronic congestion (heart disease); acute and chronic nephritis; infarction; tumor formation; tuberculosis; calculus; parasitic invasion; etc. Blood from the kidney is often not immediately apparent (the urine being smoky only); it is usually intimately mixed with the urine (although there may be clots partaking of the shape of the pelvis or the ureter); blood casts, renal and pelvic epithelium are usually found in the sediment, and blood corpuscles may be missed (that is, hemoglobinuria may occur alone); irrigating the bladder results in securing a clear fluid; and local symptoms are usually referred to the region of the kidney or the testicles. Cystoscopic examination and catheterization of the ureters also may be of value in differentiating vesical and renal hematuria. The association of pus and blood is significant of disease of the pelvis rather than of the renal substance. (5) Certain general disorders, such as hemophilia, scurvy, leukemia, and certain specific infective diseases, etc.

PYURIA.

Pyuria, or pus in the urine, may be due to suppuration of any part of the genito-urinary tract, or rarely to the rupture of an abscess (pelvic, prostatic, peri-appendicular, etc.) into the urinary tract. Pus occasions a general cloudiness of the urine, and a whitish or cream-colored, ropy, or tenacious sediment, which may be distinguished from a somewhat

similar deposit of phosphates by the fact that the phosphates are whiter, less dense, and are decomposed by the addition of an acid. Microscopic examination completes the differentiation.

Etiology.—Pyuria may be due to: (1) Disease of the urethra—usually urethritis, rarely a chancre. The pus is small in amount, escapes with the urine first voided, as well as between micturition, and is associated with painful micturition, other signs of local inflammation, and the presence of the gonococcus. (2) Vaginitis, in which in addition to evidences of local inflammation, vaginal epithelium and possibly the gonococcus may be encountered. (3) Cystitis, in which the urine may be acid when voided (Bacterium coli infections), but is usually or soon becomes alkaline (Proteus vulgaris, streptococcic, staphylococcic, and other infections), and is associated with triple phosphates, bladder epithelium, an ammoniacal odor, and other evidences of decomposition. (4) Ureteritis, which is difficult (in many cases impossible) to differentiate from pyelitis, except by evidences of local inflammation. (5) Pyelitis and pyelonephritis. The pus is usually considerable in amount and associated with renal and pelvic epithelium, and in pyelonephritis may escape intermittently; the urine is usually acid in reaction (calculous disease, Bacillus tuberculosis and Bacterium coli infections, etc.), but if the pyelitis is secondary to cystitis the urine may be alkaline.

ANOMALIES OF FORM AND POSITION OF THE KIDNEY.

The kidneys are subject to several malformations and malpositions of clinical interest. One or both kidneys may be absent; one may be hypoplastic and the other hyperplastic; there may be a supernumerary kidney; and the two kidneys may be fused—horseshoe kidney, disk-shaped kidney.

There may also be anomalous ureters (double or triple—unilateral or bilateral), and anomalous bloodvessels (of etiological significance in hydronephrosis). These anomalies acquire clinical interest in connection with contemplated operations on the kidney; the presence and the functional capacity of its fellow should always be determined before an operation on a kidney is undertaken, especially an operation that may comprise removal of the organ. The malpositions of the kidney (movable kidney, floating kidney, nephroptosis) are discussed in connection with visceroptosis (page 685).

CIRCULATORY DISTURBANCES OF THE KIDNEYS.

Active Congestion.—Active (acute) congestion is rare without concurrent exudation; that is, there is no sharp dividing line between acute congestion and acute nephritis. Acute congestion has been attributed

to sudden contraction of the peripheral bloodvessels induced by exposure to cold, and to increased functional activity, such as may occur in one kidney if the ureter of its fellow be blocked by calculus, clot, etc., or if the organ itself be removed. Otherwise the causes of acute congestion are the causes of acute nephritis. The lesions, except in that they disappear with removal of the exciting cause, are not distinguishable from those of the mild grades of acute toxic or degenerative nephritis.

Passive Congestion.—Passive (chronic or mechanical) congestion of the kidneys is usually part of the general venous stasis that accompanies chronic cardiac disease (myocardial, endocardial, or pericardial); it may occur also in the event of obstruction to the return circulation from the renal veins by pressure (tumor, aortic aneurysm, pregnant uterus, ascites), angulation (nephroptosis, kyphosis, etc.), or thrombosis or embolism of the renal vein or veins or the ascending vena cava. The kidney is enlarged and firm; the capsule strips readily and reveals distended veins and a smooth, dark bluish-red surface. The cortex is increased in thickness; the pyramids are purplish. In the course of time the organ may lessen in size, the capsule become slightly adherent, and the surface somewhat granular, due to the gradual growth and subsequent contraction of fibrous connective tissue and consecutive destruction of the parenchyma (the congested and contracted kidney; cyanotic induration). The *symptoms* consist of oliguria (retarded circulation of the blood and lessened arterial pressure)—perhaps 200 c.c. or less in the twenty-four hours; the urine is high colored, turbid, of increased specific gravity (1022 to 1035), deposits a considerable sediment of uric acid and urates, contains a small amount of albumin (usually less than 2 parts per 1000, Esbach's test), a few hyaline (rarely cellular) casts, and a few leukocytes and erythrocytes. The *diagnosis* is easy, being based upon a recognition of the primary disorder; other evidences of general venous stasis (enlargement of the liver and spleen, œdema, dropsy, etc.), or a local factor obstructing the renal return flow; the urinary findings; and fluctuation of the oliguria and albuminuria with the functional activity of the cardiovascular system. The cyanosis and dropsy contrast strongly with the pallor and dropsy of nephritis. The *prognosis* and *treatment* are those of the primary disorder.

Thrombosis and Embolism.—Primary thrombosis is very rare; embolism is more common, and is due usually to left-sided endocarditis. The lesions consist of the common wedge-shaped anemic infarcts —which may be simple and ultimately replaced by a contracted scar, or infected and give rise to abscesses. The *symptoms* often are not obtrusive; but the *diagnosis* may sometimes be made upon the sudden onset of severe pain in the region of the kidney (without radiation down the ureters—which distinguishes it from renal colic); enlargement and tenderness of the kidney; oliguria, hematuria, and albuminuria of sudden onset and often of rapid disappearance; and in the event of the embolus being infected, chills, fever, sweats, leukocytosis (which, however, may be already present in consequence of the primary infection).

NEPHRITIS.

The following varieties of inflammation of the kidneys may be differentiated:

I. Acute nephritis.
   (*a*) Acute toxic or degenerative nephritis. (Cloudy swelling or parenchymatous degeneration of the kidney.)
   (*b*) Acute diffuse nephritis. (Acute parenchymatous, exudative, catarrhal, desquamative, glomerular, tubular, or hemorrhagic nephritis.)
   (*c*) Acute suppurative nephritis.
   (*d*) Acute interstitial non-suppurative nephritis.
   (*e*) The kidney of pregnancy.

II. Chronic nephritis.
   (*a*) Chronic diffuse non-indurative nephritis. (Subacute and chronic parenchymatous, exudative, catarrhal, desquamative, glomerular, tubular, or hemorrhagic nephritis; the large white kidney; the large red, mottled, or variegated kidney.)
   (*b*) Chronic diffuse indurative nephritis.
      1. Secondary chronic indurative (or interstitial) nephritis. (Late stage of chronic parenchymatous nephritis; the small white kidney; the white granular kidney; the secondarily contracted kidney.)
      2. Primary chronic indurative (or interstitial) nephritis. (The red granular kidney; the gouty kidney; the primarily contracted kidney; the genuine contracted kidney.)
      3. Arteriosclerotic nephritis. (The senile kidney.)

Strictly speaking, the so-called toxic or degenerative nephritis is not a true inflammation; but it is due to factors which if prolonged or of increased virulence result in inflammation, it is often associated with more or less active congestion and slight exudation, and it is the important lesion in well-developed acute nephritis, so that it is not only wise to include it among the acute nephritides, but practically it is virtually impossible to exclude it therefrom: it represents an early stage, a minor grade, or a part of acute nephritis. Acute suppurative nephritis will be discussed under a special heading; acute interstitial non-suppurative nephritis is largely of pathological, of little clinical, interest; the kidney of pregnancy is a peculiar type of obvious etiological relationships.

Practically, therefore, in the great majority of acute cases the physician has to deal with what may be best described as acute diffuse nephritis, or simply acute nephritis; or with an acute exacerbation of chronic nephritis. The chronic nephritides can be sharply divided into: (1) Those that follow subsidence of the acute process, or perhaps begin subacutely or insidiously, and are characterized by predominant, but not exclusive

changes in the parenchyma (chronic diffuse non-indurative, or parenchymatous, nephritis); and (2) those in which the chief lesions occur in the interstitial tissue (chronic diffuse indurative, or interstitial, nephritis), but in which the renal lesions, diagnostically, prognostically, and therapeutically, may be neither primary nor of the greatest importance (cardiovascular-renal disease). The chronic diffuse, non-indurative, or parenchymatous nephritis is often subject to acute exacerbations, and should the patient survive for eighteen months or more, sclerotic or cicatricial lesions may develop, and a form of so-called mixed nephritis (secondarily contracted kidney) may ensue.

ACUTE DIFFUSE NEPHRITIS.

(*Acute Parenchymatous Nephritis; Acute Bright's Disease.*)

Etiology.—Acute nephritis may occur at any age, but is most common before the fortieth year and in males (excluding the kidney of pregnancy). Rarely it seems to follow directly upon exposure to inclemencies of the weather, especially coldness and dampness (said to cause reflex congestion of the kidneys, to favor the retention of excrementitious substances, to produce a hemolysin analogous to that which occurs in certain cases of paroxysmal hemoglobinuria, etc.); these cases usually occur in alcoholic subjects. Otherwise, acute nephritis is due to autogenous or exogenous infection or intoxication, and it is commonly a secondary process. The infectious causes comprise the infectious diseases, especially scarlatina, diphtheria, pneumonia, typhoid fever, influenza, cholera, yellow fever, smallpox, the pyococcic infections, etc. In most of these cases the renal lesions are due to toxemia and the excretion of toxins, rather than to bacteremia; the bacteremia of septicopyemia is likely to result in suppurative lesions (suppurative nephritis). The etiological intoxications comprise (in addition to the toxemias of the infectious processes): (1) Autogenous metabolic poisons, associated with pregnancy, anemia, gout, diabetes, jaundice, hemoglobinemia, burns, diseases of the skin associated with lessening of the cutaneous functions, etc.; and (2) exogenous poisons, such as spices, alcohol, ether, chloroform, turpentine, cantharides, salicyl compounds, carbolic acid, minerals, mineral acids and salts, etc. In anemic and mild toxemic states degenerative lesions (toxic degeneration of the renal epithelium) are common; in severe toxemias and in bacteremias degenerative and inflammatory lesions are likely to occur.

Pathology.—In mild cases, the kidney may be slightly (if at all) increased in size, and somewhat opaque (toxic degeneration or cloudy swelling); in more severe cases it is perhaps somewhat congested. In well-developed acute nephritis it is increased in size, the capsule is tense, strips readily, leaving a smooth surface—which in severe cases is dark reddish brown in color (hyperemic or hemorrhagic kidney); in less severe cases and in the subsiding stages of severe cases it is pale

grayish or grayish red in color (anemic kidney); in other cases it exhibits a combination of these (mottled or variegated kidney). On section the organ is usually soft, opaque (as though cooked), œdematous, friable, and drips blood; the cortex is swollen and the normal striations obscured; the pyramids are usually much congested. The toxins sometimes exhibit a selective action, in one case implicating especially the epithelium of the glomeruli, in another the epithelium of the convoluted tubules, in still another the connective tissues. In scarlatina, for instance, the glomeruli are likely to be especially involved (glomerulonephritis), and may appear as distinct, minute, pale grayish or dark reddish (hemorrhagic) points. Microscopically, the lesions may be confined to the parenchyma (toxic degeneration), or they may implicate all the tissues (acute diffuse nephritis), or they may be especially conspicuous in the interstitial tissue (acute interstitial suppurative or non-suppurative nephritis). The parenchymatous lesions consist of cloudy swelling (common in most of the acute infections); fatty degeneration (common in the severe infections, in certain intoxications such as phosphorus poisoning, and in severe anemias); hydropsical degeneration and complete necrosis (which occur in the severest infections, such as cholera, and in severe intoxications, such as cantharidal and mercurial poisoning, etc.). In addition, in the event of inflammatory lesions, desquamation of the renal epithelium commonly occurs (desquamative nephritis), and may be excessive. Here and there the lumen of the tubules or of Bowman's capsules may be obstructed or distended by swollen and desquamated epithelium, coagulated, finely granular albumin, fat granules, erythrocytes, leukocytes, tube casts, and detritus. Special implication of the tubules or of the glomeruli suggests the terms tubular and glomerular nephritis, respectively. The interstitial tissues in the merely toxic degenerative cases may reveal no lesions whatever; in the more frank or severe cases, dilatation and overfilling of the bloodvessels, swelling and œdema of the intertubular connective tissue, and cellular exudation and proliferation occur. The lesions, as a rule, are focal rather than widespread, and are most marked in the cortex. In some cases hemorrhages occur into the tubules, within Bowman's capsules, or in the interstitial tissue (hemorrhagic nephritis); in other cases the cellular exudation consists largely of polynuclear leukocytes (suppurative nephritis), and in other cases of Unna's large mononuclear plasma cells (acute interstitial non-suppurative nephritis).

The *kidney of pregnancy* (to be distinguished from nephritis antecedent to the pregnancy and from an ordinary nephritis, due to the common etiological factors developing in a pregnant woman) develops especially in young primiparæ, in twin pregnancies, and during the latter half of pregnancy. Its nature is not well understood, although it is probably toxic in origin, the consequence of the maternal kidneys being obliged to serve as emunctories for both the mother and the fœtus; it has been attributed also to perversion of the detoxifying function of the liver and of the thyroid, to a toxin elaborated by the syncytium (syncy-

tiolysins), to bacterial infection and intoxication, to increased intra-abdominal pressure exerted especially on the renal veins, the ureters, or the cœliac ganglion (occasioning renal anemia in consequence of reflex contraction of the renal arteries, etc.). The kidney varies in appearance in different cases; it may not be distinguishable from the more common acute diffuse nephritis; it is enlarged, the capsule strips readily, revealing a smooth, pale yellowish or mottled surface. The microscopic lesions are largely degenerative, involving the parenchyma; the lesions (inflammatory) in the interstitial tissue are comparatively slight.

Symptoms.—Toxic or degenerative nephritis usually runs its course without noteworthy symptoms; the general manifestations of the initiating infection or intoxication may be not in the least disturbed, and the renal condition is recognized only by examining the urine—which exhibits the characteristics of so-called febrile urine. It is lessened in amount, turbid, of high color and increased specific gravity (1022 to 1030), and highly acid; it contains an excess of solids and a trace or a small amount of albumin (febrile albuminuria), and, depending upon the concentration, it deposits more or less sediment that contains a few leukocytes and perhaps a few hyaline tube casts. With the subsidence of the causative infection or toxic process, the renal conditions and the urine usually revert to the normal; in severe cases, however (cholera, cantharidal and mercurial poisoning, etc.), anuria may develop and soon lead to a fatal issue.

Acute diffuse nephritis may develop insidiously (as in the infectious diseases) or suddenly (as after a debauch and exposure to coldness and dampness, severe intoxications, etc.); in either event its existence is usually revealed by œdema or changes in the urine, or both; rarely by severe uremia (coma, convulsions, etc.). Œdema (due probably to toxic alterations of the endothelium of the bloodvessels and capillaries, as well as changes in the chloride metabolism, sodium chloride retention) is a characteristic sign, and although not invariably present, may be detected by careful examination in some cases in which it is thought to be absent. It may develop slowly, the patient gradually becoming swollen and pale; or it may come on suddenly within twenty-four hours of the onset of the disease (especially after a debauch, in scarlatina, etc.). It varies much in different cases; it usually develops coincidently with, rarely before, the oliguria, but it bears no relationship to the amount of urine or of the albuminuria. Usually it develops first in the eyelids, then in the face, about the ankles, in the hands, legs, scrotum, and loose, areolar, and dependent portions of the body; finally, it may become universal (anasarca), involving even the serous sacs (which rarely may exhibit collections of fluid before the subcutaneous tissues). Œdema of the meninges may occur (and may cause coma, hemiplegia, etc.); œdema of the mucous membranes, except perhaps the glottis, palate, and conjunctiva, is uncommon; and œdema of the lungs may develop, especially in cases proceeding to a fatal issue.

Diminution in the amount of urine may be the first symptom to attract

attention; the daily amount may be 300 c.c. or less; in some cases complete anuria supervenes and may lead to a fatal issue. The urine is acid in reaction, turbid, highly colored (smoky, rarely bright red, from the presence of more or less blood), and of increased specific gravity (1024 to 1030); albuminuria (the albumin of the blood serum, serum albumin, and globulin; sometimes also nucleo-albumin) is constant—0.3 to 1 per cent. (5 to 10 grams) daily; the total output of urea and other nitrogenous substances is lessened (although the percentage of urea may be increased); the purin bases may be increased. The usually abundant (sometimes obviously hemorrhagic) sediment consists of erythrocytes (and blood pigment), leukocytes, renal and vesical epithelium, hyaline, granular, and cellular (epithelial, leukocytic, erythrocytic) casts, compound granule cells, crystals (especially uric acid and oxalates), cell detritus, bacteria (not always extraneous contamination), etc. The molecular concentration or osmotic pressure of the urine is usually reduced (hyposthenuria), so that the freezing point (cryoscopy) is 1° or less than 1° C. (instead of the normal 1.3° to 2.3° C.) below that of distilled water (0° C.). The severity of the urinary changes (amount of albuminuria, number and character of the casts, presence or absence of blood, etc.) is significant of the severity of the renal changes.

The general symptoms are not characteristic; local symptoms are often in abeyance, although rarely there may be lumbar pain or tenderness, frequent micturition, vesical tenesmus, etc. If the disease develops suddenly (as after a debauch) or in children there may be fever (101° to 103° F.), associated or not with chilliness, nausea and vomiting, headache, and, in children, convulsions; developing during the course of one of the infectious diseases there may be (but not necessarily) an added increment of fever. Fever, if it occurs, may last for several days or a week. The pulse is usually full and of increased tension, often slowed in the early, and perhaps accelerated in the later, stages. Accentuation of the aortic second sound and some hypertrophy of the left ventricle may develop after the lapse of four to six weeks; perhaps earlier in children. Epistaxis is a suggestive symptom in some cases. Dyspnœa (due to bronchitis, bronchopneumonia, œdema of the lungs, hydrothorax, hydropericardium) may occur, especially in serious cases. The appetite is poor, vomiting may ensue (its persistence indicates the early advent of more ominous uremic symptoms), constipation may be present, but diarrhœa is not unusual. Loss of strength and flesh are common, but emaciation is usually concealed by œdema. Uremia develops and is progressive; the blood is hydremic and of lessened specific gravity, and shows the common changes of secondary anemia (oligocythemia with lowered color index—disproportionate oligochromemia); the leukocytes are usually normal, but there may be a moderate leukocytosis (16,000 to 22,000), especially in severe cases of acute hemorrhagic nephritis, in uremia, and in complicating infections. The blood becomes toxic, and its freezing point higher than normally. Severe uremic manifestations (coma or convulsions) may

develop at any time, although in acute nephritis, as a rule, they occur only when there is marked oliguria.

Diagnosis.—The diagnosis may be suggested by the history of the case and the onset of œdema, but it is made by an examination of the urine. Frequent examination of the urine, therefore, should be made in all cases in which the likelihood of the development of nephritis is even remote, but especially in infections, intoxications, coma, convulsions, and pregnancy. Whether the renal lesions constitute an acute nephritis or (as is very common) an acute exacerbation of an already existing subacute or chronic nephritis, may be determined in part by the history of the patient, and in part by an examination of the urine, especially the sediment.

Prognosis.—The mild degenerative lesions usually subside with the initiating infection or intoxication; severe lesions (cholera, yellow fever, phosphorus poisoning, corrosive sublimate poisoning, etc.) may contribute or lead more directly to a fatal issue (anuria, uremia). Acute diffuse nephritis of mild or intermediate grade results in recovery in about 50 per cent. of the cases within four to six weeks; an additional percentage of cases pass into the subacute or chronic stage. The prognosis is bad in the event of extreme anasarca, marked uremia that does not respond promptly to treatment (recovery may ensue), and complications (secondary infections, œdema of the lungs, bronchopneumonia, serous membrane transudates and inflammations, etc.). The kidney of pregnancy may lead to fatal termination at almost any time, but the dangers are much minimized by judicious treatment, and recovery usually follows parturition.

Treatment.—In infections the likelihood of nephritis developing may be prevented by the avoidance of exposure to coldness and dampness; the use of suitable personal and bed-clothing; the non-use of irritating food and drink; diluting the toxins and other excrementitious substances to be eliminated by the kidneys; and promoting the functional activities of the skin and intestines. It is said that an imminent attack of nephritis manifested by hematuria and increased arterial tension may be averted or mitigated by an active cathartic—compound jalap powder or a saline cathartic.

When acute nephritis has developed, we must secure for the patient rest, warmth, and a suitable diet; the rest must be absolute and in bed until all evidences of the acute manifestations have disappeared. He should rest between blankets and wear light flannels—the absence of sheets lessens the likelihood of chill and the flannels promote the functional activity of the skin. The diet should be for a time at least as non-nitrogenous as possible, but of sufficient caloric value. Milk is the most suitable, and if possible should constitute the sole diet for four to six weeks; it may be diluted one-third with lime water (which serves to decrease the acidity of the urine) or with plain hot water, or an alkaline carbonated water, or barley water, or thin oatmeal gruel. The use of bland fluids also is serviceable—pure water to which sodium bicarbonate,

potassium citrate, or sodium benzoate (5 to 10 grains, 0.3 to 0.6 gram to the ounce) may be added; weak lemonade; a dram of cream of tartar, the juice of a lemon, added to a pint of boiling water; thin arrowroot, etc.

Local depletive measures (dry cups, followed by hot linseed poultices or other forms of hot moist heat, such as compresses, or the Paquelin cautery) sometimes seem to relieve the engorgement of the kidneys; they are especially serviceable in cases that begin with local distress, hematuria, anuria, etc.

The use of diaphoretics and purgatives, by promoting the functional activities of the skin and the intestines, serve to secure some physiological rest for the kidneys and to avert the dangers that threaten from the accumulation of toxic substances in the blood. Diaphoresis is best attained by the use of hydrotherapy—the hot-water bath, the hot-air bath, the hot-vapor bath, or the hot wet pack, repeated daily in serious cases, but less frequently in less serious cases and as convalescence becomes established. The sweating thus induced may be increased by the concurrent administration of a hot drink or a diaphoretic mixture (spirit of nitrous ether, solution of ammonium acetate, etc.). In urgent cases pilocarpine also may be used—$\frac{1}{8}$ to $\frac{1}{6}$ grain (0.008 to 0.01 gram), repeated perhaps in a half-hour if necessary; but it must be employed with caution, only when necessary, and it must be avoided in children. Free purgation is called for in all cases, and is best attained by the use of saline cathartics, compound jalap powder, etc. Calomel should be avoided, in the early stage assuredly, since it has a diuretic action attained by provoking renal congestion. Similarly, all stimulating diuretics are contra-indicated in the early stages. Water is the best diuretic; it flushes the kidneys, facilitates the removal of the products of inflammation and desquamation, lessens the acidity of the urine, and dilutes the toxins in the blood and those passing through the kidneys. The bland alkaline diuretics already mentioned may be added to the water. In the later stages the more stimulating diuretics, such as digitalis, strophanthus, caffeine, cocaine, diuretin, theobromine, etc., may be serviceable.

Special symptoms sometimes demand special treatment. Headache may require the use of bromides. Should severe uremic manifestations (coma, convulsions) supervene, venesection should be performed, and ten to twenty ounces of blood abstracted. In an asthenic subject the volume of blood removed may be replaced by an equal volume of physiological saline solution (half or one-fourth strength in the event of œdema). Convulsions not thus relieved may be controlled by a few whiffs of chloroform; by chloral (15 to 30 grains, 1 to 2 grams) and potassium bromide (60 grains, 4 grams) by the rectum; or by a single hypodermic injection ($\frac{1}{4}$ grain, 0.015 gram) of morphine. In some cases lumbar puncture has been efficacious in relieving the convulsions and the coma; the cerebrospinal fluid is often under increased pressure in these cases. The bowels should be opened immediately and freely, by an enema and

by the use of croton oil (2 drops) in olive oil on the back of the tongue, or elaterium (¼ grain, 0.015 gram). Profuse sweating also should be induced by a hot wet pack or a hot-vapor bath. Vomiting and diarrhœa (uremic phenomena) often subserve a useful purpose of elimination, and should not always be immediately checked. Vomiting, if exhausting, should be met with counterirritation to the epigastrium, and bismuth, cerium oxalate, creosote, carbolic acid, dilute hydrocyanic acid, sips of very hot water, cracked ice, etc. Anasarca that does not yield to measures already mentioned may require small incisions into the skin or the use of Southey's tube; often, however, it can be markedly, if not altogether relieved by the use of a salt-free (or salt-poor) diet (bread, meat, potatoes, eggs, etc., cooked and eaten without salt). Transudates in the serous cavities should be tapped. Pulmonary œdema, bronchopneumonia, and other complications are to be treated on general principles. During convalescence care should be taken to avoid chill and possible relapse (avoidance of exposure; use of flannels, etc.). The diet should be gradually augmented; first, farinaceous foods, then light vegetables, non-acid fruits, meats, etc. Anemia and weakness suggest the use of iron (liquor ferri et ammonii acetatis) and strychnine, but iron should not be employed unless there is anemia, nor until the acute manifestations have subsided.

ACUTE SUPPURATIVE NEPHRITIS.

(*Acute Hematogenous Infection of the Kidney.*)

Etiology.—Infection of the kidney leading to suppuration may be provoked by staphylococci, streptococci, pneumococci, gonococci, colon bacilli, typhoid bacilli, tubercle bacilli, etc. Infection may occur (1) by way of the blood stream; (2) by way of the ureter—pyelonephritis (page 788); or (3) by contiguity or continuity of tissue—perinephritic abscess (page 792). The hematogenous infections constitute a special form of nephritis. The condition may be apparently primary, and of traumatic or undiscoverable cause; or it may be secondary, and follow one of the infectious diseases (typhoid fever, diphtheria, etc.); septicopyemia or a single suppurative focus (furuncle, phlegmon, osteomyelitis, erysipelas, etc.); or some intestinal infection, especially in children.

Pathology.—The lesions consist of foci of suppuration in varying stages of development—from mere leukocytic accumulations to well-marked necroses and small, usually miliary abscesses; these are usually situated in the cortex, and although they are usually bilateral, they are often unilateral.

Symptoms.—The symptoms consist of the antecedent disorder (infectious disease, localized suppuration, etc.), to which, at the time of infection of the kidney, sudden increase of fever, chills or chilliness, perhaps sweating, and leukocytosis are added; and to these also local-

izing signs, such as abdominal pain, tenderness over one or both kidneys anteriorly, and tenderness and muscular rigidity at one or both costovertebral angles posteriorly (especially significant if unilateral). The urine, as a rule, is somewhat reduced in amount, and contains albumin, pus, blood, and casts. In some severe hyperacute cases the onset is sudden and severe, prostration is marked, fever is high (104° to 106°), and the general picture corresponds with that of general septicopyemia and obscures the local phenomena referable to the kidney. In less severe or subacute cases there are more or less protracted chills, fever, sweats, leukocytosis, and unilateral costovertebral tenderness and muscular rigidity.

Diagnosis.—The diagnosis is often uncertain, but may often be made by attention to the etiological factors, the local signs, the urinary changes, and the absence of any vesical or renal pelvic condition that might cause urogenous infection of the kidney tissue (cystopyelitis and pyelonephritis).

Prognosis.—The prognosis depends upon the etiological factors and whether the condition is unilateral or bilateral.

Treatment.—The treatment, in general, is that of septicopyemia. The local lesions may be favorably influenced by bacterins corresponding to the infection. In unilateral infections not otherwise remediable, resort should be had to surgical intervention.

CHRONIC DIFFUSE NON-INDURATIVE NEPHRITIS.

(*Subacute and Chronic Parenchymatous Nephritis; Chronic Bright's Disease.*)

Etiology.—Chronic diffuse non-indurative (or parenchymatous) nephritis is most common in young male adults between the ages of twenty and forty years; but it is not rare in women, and in children especially after scarlatina. It may follow acute nephritis, and has then a like etiology and pathogenesis. Perhaps in most cases it develops insidiously, subacutely, or chronically, and is attributed to continued or repeated exposure to the inclemencies of the weather, insanitary surroundings, overindulgence in alcohol, or infective or toxic agents insufficient in amount or virulence to cause acute nephritis (malaria, tuberculosis, syphilis, prolonged suppuration, in which event it may be associated with amyloidosis, etc.). Although the course is chronic, acute exacerbations are common; indeed, many cases of so-called acute nephritis are likely acute exacerbations of previously unrecognized subacute or chronic nephritis.

Pathology.—The lesions are not essentially different from those of acute nephritis, except in the addition of secondary alterations, the development of which requires time. Two types of kidneys occur—the large white (more properly pale or yellow) kidney, and the large red, mottled, or variegated kidney. Both are enlarged (the yellow more so than the red); the capsule strips readily, revealing a smooth surface—

pale yellowish, or mottled grayish yellow, or reddish yellow, in the one; and grayish red or mottled (commonly of pale and dark areas) or very dark reddish brown or hemorrhagic, in the other (chronic hemorrhagic nephritis). The cortex is swollen and pale, mottled, or dark red, and hemorrhagic. Should the patient survive eighteen months or more, the lesions of indurative nephritis gradually develop (secondarily contracted kidney). Microscopically, the glomeruli exhibit proliferative and degenerative changes, involving the epithelium and the endothelium, similar to those of acute nephritis; the changes in the tubular epithelium are especially marked in the convoluted tubules, and are similar to those of acute nephritis, fatty degeneration, however, being conspicuous (fatty degeneration and anemia being largely responsible for the pallor of the kidney). The changes in the interstitial tissue are similar to those of acute nephritis, usually, however, less active, more advanced; in some cases there is considerable hemorrhage (acute exacerbations; chronic hemorrhagic nephritis). In the course of time the cellular exudate becomes converted into newly formed fibrous connective tissue, which by contracting leads to atrophy of the glomeruli and tubules (secondarily contracted kidney).

Symptoms.—The disease may develop out of acute nephritis—pallor, dropsy, and albuminuria persisting. Usually it develops insidiously. The patient may complain of gradual failure of health, loss of flesh and strength, headache, pallor, indigestion, with periodic attacks of nausea and vomiting, etc.; or swelling of the eyelids, feet, or ankles; or cloudiness and lessened amount of urine may attract his attention; or albuminuria may be detected accidentally (examination for life insurance, etc.). As in acute nephritis, the characteristic signs consist of œdema (which rarely may be absent) and of changes in the urine. At first the œdema is usually slight, but it varies much in different cases; it partakes of the characteristics already mentioned in connection with acute nephritis, and having disappeared is likely to recur. The urine is lessened in amount (usually 300 to 700 c.c.), acid in reaction, turbid, of varying color (depending upon the concentration and the amount of contained blood), of increased specific gravity (1018 to 1025), contains considerable albumin (1 to 3 per cent.; $\frac{1}{2}$ to $\frac{3}{4}$ by bulk after the heat and acid test; 15 to 30 grams in the twenty-four hours); the excretion of solids is deficient; the sediment is usually abundant, and consists of: erythrocytes and leukocytes; epithelium from the uriniferous tubules, the renal pelvis, and the bladder; hyaline, granular, and cellular (epithelial, fatty, leukocytic, and sometimes erythrocytic) casts; waxy casts (in associated amyloid disease); compound granular cells, fat droplets, detritus, bacteria, etc. The general symptoms are much like those mentioned in connection with acute nephritis. Debility and anemia are usually marked, the anemia seemingly bearing some relationship to the rapidity of the renal lesions. Chronic uremic (especially nervous and gastro-intestinal) symptoms are common and persistent, although subject to remissions and exacerbations; acute uremia is less common

than in acute nephritis and chronic indurative nephritis. The blood pressure is usually increased, and hypertrophy of the heart and arteriosclerosis often supervene. Albuminuric neuroretinitis is quite common.

Diagnosis.—The diagnosis is suggested by the history of the case, pallor, and œdema, but it must be based upon an examination of the urine. Much fat (fatty casts, free fat droplets, etc.) suggests the pale kidney; blood, the red or mottled kidney—an acute exacerbation. The amyloid kidney is suggested by the etiological factors of amyloid disease, enlargement of the liver and spleen, relatively slight œdema, relatively few casts (and these hyaline, waxy, and granular), little or no hypertrophy of the heart, and the absence of uremia and of neuroretinitis. The advent of secondary renal contraction leads to symptoms that will be mentioned under chronic indurative nephritis.

Prognosis.—The duration of the disorder commonly varies from several months to two years or thereabouts; remissions and exacerbations are common; some patients gradually developing a secondarily contracted kidney may survive for a number of years. The outlook however, is always gloomy, since recovery is extremely rare. The prognosis is the worse the greater the degree and persistence of albuminuria, œdema, and uremic manifestations, the less the excretion of urinary solids, and the more marked the cardiovascular changes, the neuroretinitis, and other complications.

Treatment.—The treatment, hygienic, dietetic, and medicinal, is similar to that of acute nephritis. From time to time a strict milk diet is valuable. In other cases with marked œdema benefit accrues from a salt-poor diet; at all events, as little salt as possible should be given. The œdema may also be favorably influenced and perhaps the tendency to uremic manifestations lessened in some cases by Basham's mixture; or by Trousseau's diuretic wine:

℞—Powdered digitalis	1 dram	4 0
Powdered squill	½ dram	2 0
Juniper berries	5 drams	20 0
Potassium acetate	2 drams	8 0
White wine	6 ounces	200 0—M.

S.—One tablespoonful (15 c.c.) 3 or 4 times daily.

or by Grainger Stewart's mixture:

℞—Potassium acetate	2 drams	8 0
Infusion of digitalis	3 ounces	100 0
Infusion of scoparius	3 ounces	100 0—M.

S.—One tablespoonful (15 c.c.) 3 or 4 times a day.

Strontium lactate, diuretin, theobromine, sparteine, etc., may render service from time to time. Renal decapsulation has been practised with good results in some cases. The treatment of uræmia is mentioned on page 778.

CHRONIC DIFFUSE INDURATIVE NEPHRITIS.

(Chronic Interstitial Nephritis; Arteriosclerotic Kidney; Chronic Bright's Disease;) Cardiovascular-renal Disease.)

Etiology.—Chronic diffuse indurative (or interstitial) nephritis may be primary or secondary. The primary condition occurs especially after the fortieth year, but it may occur even in children. It is twice as common in men as in women. The disease is largely the result of the exigencies of modern life: the hurry and the cares, the worries and the anxieties of modern life, the nervous tension and the mental strain inseparable from large undertakings, heavy financial responsibilities, and high society. An irregular mode of life, overeating of rich and highly seasoned food, excessive indulgence in alcohol, and insufficient muscular exercise tend to increase blood pressure and to pervert metabolism, the products of which being eliminated by, irritate and cause progressive deterioration of, the chief emunctories, the kidneys. In some cases the disease is directly attributable to certain autogenous and exogenous poisons, such as alcohol, gout (gouty kidney), lead, etc.; and sometimes the disease seems more or less related etiologically to certain infections, such as syphilis, malaria, etc. The causes of the disease in general are those of arteriosclerosis, which is apparent in the separation of a special type of the disease (arteriosclerotic kidney) believed to originate in the bloodvessels rather than in the kidneys; in another class of cases the lesions appear to be merely those of advancing age (senile kidney). The phenomena in most cases are correctly interpreted by recognizing that the disease is not a disease of the kidneys alone, perhaps not even primarily, but a disease of the cardiovascular system and the kidneys (cardiovascular-renal disease). Secondary chronic diffuse indurative nephritis develops, as a rule, secondarily to the large white kidney; its etiology, therefore, is that of the antecedent condition.

Pathology.—In the primary (and the arteriosclerotic) process the kidney is much reduced in size (both together may weigh less than 50 grams); the capsule is thickened and markedly adherent, and when removed reveals a dark reddish brown, markedly granular surface, containing, as a rule, a variable number of small cysts; the organ is firm hard, and dense; the cortex is much reduced in thickness (to 2 mm.). The arteries show more or less sclerosis. Microscopically, the conspicuous change is an overgrowth of cicatricial fibrous connective tissue with associated (consecutive) atrophic and degenerative changes in the parenchyma. The fibrosis is especially marked about Bowman's capsule and the small arteries—whence it spreads to the adjoining tissues; the Malpighian bodies ultimately may be represented by a functionless, roundish, hyaline mass, and the small arteries may become completely fibrosed and their lumens occluded. Obstruction and occlusion of uriniferous tubules give rise to the formation of small cysts. In the arteriosclerotic kidney the lesions are believed to

originate in the bloodvessels, but it is often quite impossible to distinguish between the two forms of the disease—if there really be two forms. The secondarily contracted kidney is sometimes slightly enlarged, sometimes normal, and sometimes somewhat reduced in size; it is firm and hard; its capsule is thickened and adherent, and when removed reveals a granular pale yellowish or mottled yellowish-red surface, containing a variable number of small cysts; the cortex is thinned. Microscopically, to the lesions of the large white kidney are added overgrowth and cicatrization of connective tissue, more or less fibrosis and obliteration of the glomeruli, arteriosclerosis and obliterating endarteritis, occluded and distended uriniferous tubules (cysts), etc. In addition, in all forms of chronic indurative nephritis there is more or less widespread arteriosclerosis, and hypertrophy, and later, dilatation of the heart (page 437); and perhaps fibrosis elsewhere in the body.

Symptoms.—The symptoms of primary chronic indurative nephritis develop as insidiously as do the lesions in the kidneys and the cardiovascular system. The disease may exist for years entirely devoid of symptoms; it may be detected accidentally in a routine examination of the urine for life insurance or other reason; or it may give rise to an extremely variable set of initial symptoms that are likely to be attributed to some organ other than the kidneys. Thus, the patient may present himself to his physician with the idea that he is suffering with diabetes on account of the large amount of urine that he voids; or he may complain of dimness of vision, the consequence of albuminuric neuroretinitis; or of one of the many manifestations of chronic uremia, such as dyspnœa or asthma; or of gastro-intestinal derangements, such as gastric catarrh, gastric hemorrhage, or diarrhœa; or of headache, hemicrania, tinnitus aurium, vertigo, neurasthenia, pains in the muscles or joints, or eczema; or palpitation of the heart and precordial distress; or, the patient in apparently good health, may suddenly develop a severe and even fatal attack of cardiac failure, or he may pass into uremic coma or convulsions, or he may suffer an apoplexy and die.

Variable as are the modes of onset, the diagnosis of the disease depends upon an examination of the urine and of the cardiovascular system. The urine is increased in amount—1800 to 4000 c.c. daily—whence the common complaint of frequent micturition and sometimes of increased thirst; it is acid in reaction, pale in color, and of low specific gravity (1002 to 1015); it contains a slight amount of albumin—usually only a trace, and at times no albumin at all. Albumin may be absent for days and even weeks at a time; it may be missed in the early morning urine and be present in that voided later in the day; and it may be present only after exercise, emotional and other excitement, or after eating, especially after eating proteins. The urine deposits a slight flocculent sediment that contains a few, sometimes a very few (often very narrow), hyaline and pale granular casts, and a few epithelial cells, mostly from the bladder. The solids of the urine are usually diminished.

The characteristic cardiovascular signs consist of increased blood pressure (an early and valuable sign), arteriosclerosis, accentuation of the aortic second sound, and hypertrophy of the heart, especially the left ventricle (sometimes obscured by co-existing emphysema). Although these cardiovascular signs may be found in all forms of Bright's disease (but not in all cases), in none of the forms do they so dominate the clinical picture as in primary chronic interstitial nephritis. So long as the nutrition of the heart is well maintained there may be no symptoms, although for months or years dyspnœa and palpitation of the heart may be provoked by exertion or excitement. As the nutrition of the heart begins to lag behind its enlargement, degeneration and dilatation ensue and symptoms of embarrassed circulation become ascendent—cardiac asthma, congestion and œdema of the lungs, bronchitis, gastro-intestinal disturbances, generalized œdema and effusions within the serous cavities, and alterations in the urine: it lessens considerably in amount and becomes of high color and increased specific gravity; albumin increases in amount and casts become more numerous; dark granular, epithelial, and even blood casts and free erythrocytes may be encountered. The symptoms referable to the heart and lungs, at first transitory and nocturnal, become permanent; the blood pressure lessens; the aortic second sound becomes less accentuated; with embarrassment of the right heart the pulmonary second sound becomes at first accentuated, and later fails; signs of dilatation, and gallop rhythm, always of serious moment, supervene; and if relief is not afforded, death ensues with cardiac failure or uremia, or both, dominating the final scene.

In some cases the first suggestive symptom may be œdema of the lungs or of the larynx; the dyspnœa due to one or both of these may be augmented by hydrothorax; Cheyne-Stokes breathing may ensue, and in coma the patient may die. Or the first symptom may be violent headache—due either to uremia, cerebral congestion, or cerebral anemia. The headaches are occasionally relieved by epistaxis, which is sometimes frequent and sometimes severe. Drowsiness and other nervous manifestations of chronic uremia may be associated with the headache, and frequently presage the onset of severe, acute, and fatal uremia. In some cases psychoses are prominent; in other cases neuralgias or hemorrhages into the brain substance or into the meninges may occur. Hemorrhages into other structures, especially the stomach, intestine, skin, conjunctivæ, lungs, etc., have been observed. Severe and uncontrollable vomiting and severe and exhausting diarrhœa are not uncommon. In other cases the disease is manifest by intolerable itching, formication, numbness of the extremities; in still other cases, by urticaria. In the late stages more or less widespread erythematous, or bullous, or desquamative skin lesions may occur. Neuroretinitis is the most important symptom on the part of the special senses; at times tinnitus aurium, possibly associated with dizziness, occurs; less frequently deafness or diplopia ensues.

Secondary chronic interstitial nephritis (the small white kidney)

is usually only with difficulty differentiated from its antecedent stage (the large white kidney). Presumptive evidence of secondary contraction of the kidney, however, is afforded by long duration of the disease (more than a year), lessening of dropsy, lessening of albuminuria, increase in the daily excretion of the urine, and decrease in the specific gravity, and the development and progression of the cardiovascular signs of chronic interstitial nephritis.

Diagnosis.—As long as cardiac compensation is maintained, the diagnosis of chronic indurative nephritis is evident from the results of an inquiry into the antecedents of the patient, the special etiological factors in the case, the course of the disease, the different clinical manifestations, and an examination of the urine and of the cardiovascular apparatus. The diagnostic importance of the cardiovascular alterations cannot be overestimated. They are among the earliest clinical manifestations of the disease, and they may be detected even in the absence of positive signs of kidney disease on the part of the urine.

It is usually a matter of extreme difficulty to distinguish clinically between primary chronic interstitial nephritis and the arteriosclerotic kidney; in some cases it is impossible, in most cases it is unnecessary. Usually the arteriosclerotic kidney exists for a long time without albuminuria, or with but the slightest trace of albumin in the urine. The disease may be suspected in elderly persons, with hypertrophy of the heart, increased arterial tension, and marked atheroma of the superficial arteries. Occurring as a manifestation of senility, it is usually of relative unimportance as contrasted with the co-existing emphysema, dementia, marasmus, etc.

When the heart begins to fail in chronic interstitial nephritis, diagnostic difficulties arise. The clinical picture in these cases much resembles that of a disorder primarily cardiac and secondarily renal. The presence of murmurs is not conclusive evidence, since they may be due to dilatation as well as to valvular disease. Diastolic murmurs, however, suggest a primary cardiac disorder, whereas albuminuric neuroretinitis suggests a primary renal disorder. In some cases the diagnosis, for a time at least, is impossible; in other cases it may be made by a careful analysis and balancing of all the features of the case, by a knowledge of the past history of the patient, and by the results of administering digitalis and other heart tonics. Following the administration of digitalis, should the patient improve materially, the urine increase in amount, and albumin and casts finally disappear from the urine, the condition has been definitely determined to have been primarily cardiac with secondary renal congestion. If, however, with improvement in the condition of the heart, albumin and casts do not disappear from the urine, one may be confident of the existence of nephritis, a conviction that may be confirmed by examining the cardiovascular apparatus now that compensation has been restored. It is scarcely likely that chronic indurative nephritis will be confounded with the so-called functional, physiological, cyclic, intermittent, paroxysmal, or adolescent albuminuria.

Prognosis.—In general the prognosis depends upon the integrity of the cardiovascular apparatus. The disease may run a course entirely latent and lead to sudden death by apoplexy, acute uremia, etc. Ordinarily, however, the course is from ten to twenty years, depending upon the habits and mode of life. When the circulation becomes embarrassed the prognosis is bad; lessening in the amount of urine, especially if associated with headache, often presages the speedy onset of acute uremia.

Treatment.—In the early stages of the disease, appreciating our inability to cure our patient, we should be alert to the dangers that threaten him (rupture of small arteries, especially of the brain, uremia, cardiac failure). He must be cautioned not to expose himself to the inclemencies of the weather, to avoid all excessive muscular exercise and all mental worry; he must have his bowels open at least once daily (alkaline mineral waters, saline cathartics, occasional blue mass pill, etc.); and he must favor the action of the skin so as to relieve the subnormal cardiovascular-renal system; warm or hot baths are serviceable, but they are contra-indicated if they cause unpleasant throbbings (increasing blood pressure). The diet is of the utmost importance, the duration of life probably depending more upon discretion in eating and drinking than upon any other factor. In many cases an exclusive milk diet for a time (four to six weeks) is desirable; and it may be repeated from time to time. In general the diet should be sufficiently varied to be attractive, it should be free from irritating qualities and readily assimilable, and the nitrogenous constituents should be relatively reduced. The meat allowance should not exceed 100 grams daily; a diet of ample fuel value should be constructed by adding a sufficiency of fats and carbohydrates; an excess of meat is said to favor the development of uremia; red and white meats are of equal value, and may be allowed in accordance with the patient's desires. As a rule, alcohol should be prohibited, although in some cases a little of the lighter wines appears to do no harm. In many cases the foregoing produces not only amelioration of the symptoms, but perhaps also arrest of the lesions; if so, other measures are not required.

When the circulation becomes embarrassed medication is called for—the most serviceable remedies being nitroglycerin and caffeine. Nitroglycerin lowers the arterial tension and enables the heart still capable of performing its work to do so unhampered by excessive resistance in the arterial tree; it is said also to favor reduction in the amount of albumin excreted, but this action is doubtful and certainly immaterial. This dose may be increased until the desired result is obtained. It is not necessary that the drug be given to the extent of producing unpleasant symptoms; sufficient reduction in the arterial tension is usually produced without this. Nor is great reduction in the arterial tension desirable; indeed, it is directly contra-indicated, since a reduction even to that of the normal tension is fraught with danger of serous effusions, and of uremia (on account of reduction in the amount of urine excreted). The nitroglycerin should be given for four to six weeks and then discontinued for a week or ten days, unless the

condition of the patient should render an earlier return to its use necessary. The nitrite of potassium or of sodium or erythrol tetranitrate may be substituted for the nitroglycerin, but they are less efficacious. Inasmuch as the nitrites interfere somewhat with the oxygenating powers of the blood, their continuous administration should not be persisted in too long. Iodide of potassium or of sodium in small doses, mercuric bichloride, and chloride of gold and sodium are still advocated; in some cases good results appear to attend their prolonged use. It is difficult, however, to estimate the amount of good that with justice may be attributed to their use. In the absence of serious cardiac debility, cardiac stimulants, such as digitalis and strophanthus, should not be given. Nor is the routine administration of iron to be countenanced. Anemia is rarely marked, and any virtues that iron may possess are outweighed by certain ill effects, such as its tendency to constipate, to produce headaches, etc. However, should anemia become marked, iron may be given in small doses. Quinine, strychnine, and arsenic are often of service in this stage of the disease, as well as earlier. Renal decapsulation is sometimes of service.

When marked cardiac debility and manifest failure of compensation develop (generalized œdema, effusions in the several serous cavities, diminution in the amount of urine, increase in the amount of albumin, etc.), digitalis and other cardiac stimulants should be given. In general, the treatment of this stage of the disease does not differ from that already recommended in connection with acute and chronic non-indurative nephritis, and from that commonly practised in cases of lack of cardiac compensation. Impending uremia, manifest by headache, restlessness, foul breath, coated tongue, vertigo, etc., may be warded off by nitroglycerin and caffeine, saline cathartics, alkaline diuretics, large amounts of water, hydrotherapeutic measures, and restriction of the diet to milk. In some cases digitalis is urgently called for. The treatment of acute uremia developing in the course of chronic indurative nephritis does not differ from that already mentioned (page 778).

CYSTITIS; URETERITIS; PYELITIS; PYELONEPHRITIS; PYELONEPHROSIS.

Etiology.—Inflammation of the bladder, the ureter, and the renal pelvis may occur singly or in combination; the inflammatory phenomena may ascend from a part of the urinary tract low down to that high up, or they may descend; and they may be acute or chronic. Although many bacteria have been described in these urinary infections, the most important are the typhoid and colon bacilli (acute and chronic infections), gonococci, staphylococci, streptococci, and pneumococci (acute infections), the tubercle bacillus (chronic infections), and perhaps also certain anaërobic organisms. Ascending (urogenous) infections of the bladder are usually preceded by urethritis (or vaginitis), commonly

gonococcic; they may result from the introduction into the bladder of infected foreign bodies (catheters, etc.); or they may be due to the transit of infectious agents (commonly the colon bacilli or the pyococci) from the rectum or other pelvic viscus directly to the bladder (transparietal route). Thence the ureters, the renal pelves, and the kidneys themselves may become infected, commonly by progressive ascending infection of the mucosa; rarely by the transit of the infectious agent through the blood stream or the lymph stream. Descending infections—the consequence of the elimination of bacteria by the kidneys (therefore hematogenous infections)—occur under circumstances similar to those that induce suppurative nephritis (page 779); they are usually due to the typhoid bacillus, the colon bacillus, proteus vulgaris, the pyococci, etc. Pyelitis may be the initial lesion, but in some cases the bacteria pass through, apparently without injuring, the pelvis, and may first set up a cystitis. As is well known, the mucosa of the urinary tract is remarkably resistant to the influence of bacteria eliminated with the urine. Apparently healthy kidneys may excrete bacteria (as in many infections, notably typhoid fever), and these may be discharged from the body, without damage to the urinary tract. Certain predisposing factors seem operative in many cases, and may determine the primary local infection—such as trauma, arteriosclerosis, diabetes, a calculus (in the renal pelvis or the bladder), enlarged prostate, urethral stricture, ammoniacal decomposition of the urine, a pelvic tumor, a pregnant uterus, the excretion of irritating drugs, etc.

Pathology.—The lesions may be catarrhal, suppurative, or ulcerative; unilateral or bilateral; and acute or chronic. In the catarrhal process there is swelling, œdema, and congestion of the mucosa, with the excessive formation of mucus; in aggravated cases considerable mucopus or almost pure pus is formed, and there is more or less desquamation and superficial erosion of the epithelium and foci of hemorrhage; or more or less widespread and deep ulceration may occur. In the chronic cases there is, in addition, more or less thickening and pigmentation. Rarely fibrinous exudations or gangrene ensue. There is, as a rule, more or less decomposition of the urine with the precipitation of urates and phosphates. When the infection extends from the renal pelvis to the kidney substance (urogenous pyelonephritis) yellowish purulent streaks are found extending from the papillæ into the medulla and the cortex, and small miliary abscess throughout the kidney, but most numerous in the cortex just beneath the surface and arranged often in groups. In the event of obstruction in the renal pelvis, the ureter, bladder, or urethra, with consequent increased intrapelvic (renal) pressure, the pelvis and calices become distended, the renal tissue more or less destroyed, and the kidney ultimately converted into an intercommunicating, many-chambered pus sac (pyonephrosis) lined by the remains of the pelvic mucosa; should this become very large, very little kidney tissue may be demonstrable (degeneration, atrophy, and replacement by connective tissue).

Symptoms.—The symptoms vary with the site and progression of the lesions; in some cases, especially in children, there may be no symptoms whatever referable to the urinary tract.

Acute *cystitis*, as a rule, is manifested by pain in the region of the bladder developing spontaneously, augmented by urination, and radiating to the urethra; strangury and vesical tenesmus; and local tenderness. Occasionally, in consequence of sphincter spasm or urethral obstruction by a plug of mucus, there may be retention (ischuria). The general phenomena consist of fever, perhaps chill or chilliness (at the beginning of acute cases), leukocytosis, headache, malaise, etc. In subacute and chronic cystitis the local and general phenomena are less marked and may be quite in abeyance. The urine when voided may be acid or alkaline depending upon the infecting organism: acid in colon-bacillus infections, and alkaline in infections by urea-decomposing organisms. Furthermore, the urine contains more or less mucus, pus, erythrocytes, albumin (proportionate to the pus and blood), bladder epithelium, perhaps particles of necrotic bladder mucosa, many bacteria, and in the event of alkaline decomposition (urea-decomposing organisms), triple phosphates, ammonium urate, etc. Cystoscopic inspection of the bladder reveals the inflammatory lesions; in the chronic process—often tuberculous ulcers. The thickening that develops in chronic cases may be palpable—above the pubes; or bimanually—vaginal (or rectal) and suprapubic. Acute cystitis lasts for several days to several weeks, and may then subside entirely, or go on to the chronic stage; this may continue for months or years, and ultimately lead to ascending infection of the urinary tract.

Ureteritis is rarely if ever an independent affection; usually it is part of, or associated with, cystitis and pyelitis. Local pain, tenderness, and palpable thickness along the course of the ureter can sometimes be made out, and cystoscopic examination of the bladder often reveals excessive inflammatory phenomena at the mouths of, and perhaps some distance into, the ureters.

Pyelitis and *pyelonephritis* are manifested, as a rule, by impairment of the general health, by more or less obtrusive symptoms of septicopyemia, and by certain local signs. In the milder cases (subacute or chronic urogenous pyelitis or pyelonephritis) fever may be mild (99° to 100°) but more or less continuous; there may be a tendency to ready sweating; slight leukocytosis is likely to be present; anemia develops and progresses. In some cases, suddenly chills, fever, and sweats supervene and the leukocytosis increases; the fever may reach a high grade (104° or more), continue for several days or a week or more, and gradually subside to the previous grade. At varying intervals, such an attack may recur (recurring type). In addition there may be local pain (often spontaneous and colicky) and tenderness in the region of the kidney (or kidneys), frequent micturition, and in the event of pyelonephrosis a variable-sized, tender, fluctuating mass in the region of the kidney.

The urine contains mucus, pus, erythrocytes, albumin (proportionate to the pus and blood), fibrinous plugs, caudate epithelium from the renal pelvis (not always recognizable) and other epithelium, and crystals, often corresponding to a calculus in the renal pelvis, but otherwise varying with the alkalinity or acidity of the urine. The amount of urine varies much in some cases, being often reduced and then suddenly increased—associated with release of an obstruction and discharge of accumulated urine, lessening in size of the palpable fluctuating tumor, and subsidence of the general manifestations of toxemia. In chronic cases there may be presistent polyuria (usually due to secondary chronic interstitial nephritis).

Diagnosis.—The diagnosis of infection of the urinary tract is easy in the great majority of cases; but some difficulty may be experienced in determining the seat and extent. Cystitis is readily recognized by vesical pain and tenesmus, strangury, and the condition of the urine; and outspoken pyelitis by pyuria (especially intermittent pyuria), pain, tenderness, and a fluctuating mass in the region of the kidney. In many cases, however, these are not obtrusive, and the diagnosis depends upon the differential interpretation of pyuria; this is especially the case in marked toxemias or infections which are common in children, during pregnancy and the puerperium, etc.—whence the importance of examining the urine in all obscure febrile conditions, particularly those without any localizing symptoms. In some cases, special importance attaches to an antecedent enlarged prostate, urethral stricture, etc. Pressure on the kidney and massage in the direction of the ureter may lead to the discharge of pus into the bladder—which may be observed by means of a cystoscope. Effort should always be made to determine the nature of the infection; the tubercle bacilli may be recovered in many cases not thought to be tuberculous. More albuminuria than can be explained by the pus and blood suggests concurrent nephritis.

Prognosis.—The prognosis depends upon the nature of the infection—tuberculous or non-tuberculous, and whether simple or associated with a calculus. In acute cases the prognosis, as a rule, is good; chronic cases may last for years.

Treatment.—Efforts should be made to prevent the infection—by the use of clean catheters, etc.; by prompt and efficient treatment of gonorrhœa, vaginitis, cystitis, etc; and when the urinary tract is damaged, by avoiding irritating drugs that must be eliminated by the kidney; etc. The developed infection should be met by bland fluids (milk, water, mineral waters) to flush the urinary tract, and urinary antiseptics, such as hexamethylenamin, salol, sandal oil, etc. In urinary acidity (colon bacillus infections), potassium acetate and citrate and sodium benzoate are of much service. Autogenous vaccines corresponding to the infecting organism are of much value in many, especially, protracted cases. Local warm applications afford relief in cystitis. In protracted cystitis irrigations with mild antiseptics should be used; or persisting ulcerations

(usually tuberculous) may require the local application (through the cystoscope) of lactic acid, etc. Urethral strictures, enlarged prostate, vesical and renal calculi, etc., should be treated surgically. Pyelonephritis and pyonephrosis also usually require surgical intervention.

PERINEPHRITIS AND PARANEPHRITIS.

(*Perinephritic Abscess.*)

Etiology.—Inflammation of the fibrous capsule of the kidney (perinephritis) and of the surrounding fibro-adipose tissue (paranephritis) may be due to trauma; to extension of inflammation from the kidney (pyelitis, pyelonephritis, pyelonephrosis), from the intestine (especially appendicitis and ulcerative processes involving the colon), from the vertebræ (tuberculosis), pleura (empyema), etc.; and occasionally it follows infections, septicopyemia, etc., the process being embolic. The disorder is most common in male adults.

Pathology.—In some cases the lesions are those of more or less well-marked inflammation with considerable inflammatory œdema; the process may subside. Usually, however, the lesions progress to suppuration, the kidney being more or less surrounded with pus, sometimes embedded in a large abscess cavity; the pus may have a fecal odor (intestinal origin). The abscess may burrow downward along the psoas muscle and appear in the groin, or upward into the pleura and lungs, and perhaps be evacuated through a bronchus; or it may perforate the intestine, the peritoneum, the vagina, etc. Rarely there is a chronic indurative perinephritis, thickening and induration of the perirenal fibro-adipose tissue, and compression of the kidney (which produces no significant symptoms).

Symptoms.—In the absence of suppuration there are no unequivocal symptoms. The phenomena of abscess formation may be insidious or abrupt in onset. In the course of one of the aforementioned etiological disorders, deep-seated pain develops in the renal region; the pain may radiate to the thigh or the testicle (which may be retracted), or be referred to or near the hip-joint; the thigh may be favored or flexed (irritation of the psoas muscle), and the spine may be immobile from contraction of the erector spinæ and other muscles of the affected side. On palpation there may be swelling of the lumbar region, with perhaps œdema of the skin (especially significant); or distinct deep-seated fluctuation may be elicited. The general signs of suppuration, chills, fever, sweats, and leukocytosis, are usually present; the urine may be clear, or reveal the evidence of a concomitant (antecedent or sequential) pyelitis or pyelonephritis.

Diagnosis.—The diagnosis, as a rule, is comparatively easy—by attention to the antecedent etiological factors and the evidences of suppuration.

Prognosis.—The prognosis depends upon the primary cause, the amount of damage done, and the amenability of the condition to opera-

tion. Cure may be effected in some cases by prompt evacuation of the abscess, but in some cases prolonged suppuration and consecutive amyloid disease, etc., ensue.

Treatment.—The treatment is essentially surgical.

HYDRONEPHROSIS.

Etiology.—Hydronephrosis, a saccular dilatation of the renal pelvis and calices, associated with the accumulation of a urinary fluid, may be congenital or acquired, and unilateral or bilateral. The congenital cases (bilateral in 65 per cent. of the cases) are due to atresia of the ureter near the renopelvic junction, to congenitally displaced kidneys, or to anomalous renal bloodvessels compressing the upper part of the ureter. The acquired cases are due to traumatic and inflammatory strictures; partial or complete obstruction by a calculus, or fibrinous, or other plug; to displacement of the kidney and angulation or twisting of the ureter; or to tumors or other lesions compressing the pelvis or the ureter from without, or the ureter at its entrance into the bladder.

Pathology.—The saccular distention varies considerably in different cases, and it may be complete or incomplete, or intermittent. The larger distentions occur when the obstruction is of slow development, incomplete, and intermittent rather than when rapid and complete; sudden and considerable intrapelvic pressure soon overcomes the renal blood pressure and inhibits the excretion of urine, whereas slow increase and intermittent reduction of the intrapelvic pressure permits of more abundant secretion of urine. The kidney and the pelvis and perhaps the ureter are more or less enlarged, irregular, saccular; the renal papillæ are flattened, the calices distended, the renal tissue atrophic and sclerosed. In many cases the kidney and the pelvis form a distinct cystic tumor with more or less thinned walls.

Symptoms.—The symptoms vary with the antecedent and associated conditions; in some cases they are inconsequential. In uncomplicated cases, such as are common in young adults, the symptoms consist of periodic attacks of sudden severe abdominal pain usually localized to the region of the kidney (upper right or left abdominal quadrant and the costovertebral angle posteriorly) and not radiating down the ureter, as a rule; these attacks are often associated with nausea and vomiting. Often a more or less large, firm, or cystic tumor may be palpable in the region of the kidney—which characteristically varies in size from time to time (change in posture, etc.), decrease in size being associated with an increase in the amount of urine (intermittent hydronephrosis). The changes in the urine are variable, and depend more upon associated conditions (inflammations, tumors, calculi, etc.) than upon the hydronephrosis itself. The intermittent passage of a large amount of clear, pale urine of low specific gravity is indicative of the intermittent type of the disorder. In the bilateral disorder, which is usually more or less permanent, the amount of urine may be below normal.

Diagnosis.—The diagnosis in uncomplicated cases is usually easy. Other forms of renal and other retroperitoneal tumors must be excluded. Pyonephrosis may be excluded by the absence of evidence of septicopyemia, and of pus from the urine (although some pus cells may be found microscopically in many cases of hydronephrosis). The acute attacks of pain simulate appendicitis, cholelithiasis, and renal calculus; ordinarily little difficulty should be experienced in excluding these.

Prognosis.—The prognosis depends entirely upon the underlying condition and its amenability to treatment.

Treatment.—The treatment is essentially surgical, although if the condition occasions little or no distress, operation need not be insisted upon. Division of anomalous bloodvessels where they cross and obstruct the ureteropelvic junction suffices in some cases to cure the condition; in other cases plastic operations on the ureter and pelvis, nephrotomy, anchoring a displaced kidney, etc., may be necessary; or complicating calculi, tumors, etc., must be removed.

NEPHROLITHIASIS.

(*Renal Calculus.*)

Etiology.—Renal calculi are most common in adult life (thirtieth to sixtieth year); and in males (75 per cent. of the cases). Their formation is said to be favored by a sedentary life and by habits in eating and drinking that favor the development of gout. Bacterial infection is an active factor in many cases.

Pathology.—Primarily urinary calculi are renal in origin in the very great majority of cases; but secondary accretions may occur in the renal pelvis, in the ureter, or in the bladder, and calculous deposits may occur in and about divers inflammatory and necrotic processes in the urinary tract—whence a distinction is sometimes made between primary and secondary calculi. Furthermore, calculi may be solitary or multiple, and unilateral or bilateral (20 to 25 per cent. of the cases). According to size one may distinguish: (1) Renal sand, fine, gritty particles; (2) gravel, concretions up to 1 to 2 mm. in diameter; and (3) calculi (or stones), varying in size from that of a pea or bean to large dendritic or coral-shaped formations that fill out even a dilated renal pelvis and the calices. Chemically the calculi may be composed of: (1) Uric acid and urates—the most common (two-thirds of the cases). Sand and gravel almost always consist of uric acid and urates; often also the small solitary or the larger dendritic stones. These are hard, reddish in color, have a smooth surface, and on section are dense and stratified; and they occur in acid urine. (2) Calcium oxalate (mulberry calculi, the second in point of frequency), hard, gray or dark in color, and with a rough surface, or studded with spines; they also occur in acid urine. (3) Phosphates (calcium and ammonio-magnesium), small, grayish in color, and soft; they are uncommon, and occur in alkaline urine;

phosphates may form a coating of other calculi. (4) Certain rarer forms, such as calcium carbonate, xanthin, cystin, indigo, and urostealiths (fatty calculi, which originate usually in the bladder). Precipitation of the urinary salts may occur in the kidney substance—so-called renal infarcts, of which the following varieties may be distinguished: (1) Uric-acid (urate) infarcts, which occur in the newborn and in gouty subjects, usually at the apices of the pyramids; (2) calcium (phosphate and carbonate, perhaps also oxalate), whitish opaque streaks, found especially in the pyramids in senility, metallic poisoning, etc.; (3) pigmentary infarcts—derivatives of hemoglobin, biliary pigment, etc.

Calculi induce various lesions in the urinary tract. In some (aseptic) cases there may be few if any lesions beyond the mere presence of the calculus; in other (also aseptic) cases there may be more or less hydronephrosis or chronic interstitial connective-tissue overgrowth with contraction and atrophy of the renal parenchyma. In infected cases there is an associated pyelitis, pyelonephritis, pyonephrosis, or perinephritic or paranephritic abscess. In unilateral aseptic cases there may be more or less hypertrophy of the opposite kidney; in infected cases there is often a descending-ascending infection of the bladder and the other kidney.

The mode of formation of renal calculi is not thoroughly understood. Roberts believes that the main factors in the formation of uric acid salts are high urinary acidity (which favors the deposition of uric acid), excess of uric acid, and deficiency of salines and of pigments. Ebstein has proved that urinary calculi are not simple conglomerations of crystalline deposits, but that they possess an organic framework, the product of pathological processes in the urinary tract, in which the crystalline deposits are embedded and held together. Schade, pointing out the fundamental error of believing that the urinary constituents exist in simple watery solution, emphasizes the importance of the colloids in maintaining solution; he states that when the crystalloids go out of solution they take with them colloids (pigment, etc.) and form concretions with a complex colloidal stroma, to which is due the well-known insolubility of the calculus as compared with a simple crystalline mass of the same chemical nature. The special colloid is believed to be fibrin or fibrinogen, a product of inflammation; which facts, in connection with the frequency of infection of the urinary tract and the finding of bacteria in many urinary calculi suggest that urinary calculi, in part at least, are infectious or inflammatory in origin (analogous to biliary calculi); in part there is perhaps an excess in the urine of the crystalloids in question or a lessened ability of the urine to hold them in solution.

Symptoms.—Nephrolithiasis may be entirely latent; a large dendritic calculus may constitute an accidental necropsy finding; or sand or gravel may be passed from time to time for years with little or no noteworthy discomfort; or oliguria or anuria may suddenly develop from ureteral obstruction; or such may be the destruction of renal tissue that uremia may come on without obtrusive antecedent symptoms.

In the majority of cases, however, the disorder is manifest by renal pain, hematuria, pyuria, and other evidences of pyelitis or pyelonephritis. The pain may be dull and aching in character in the region of the kidney, and more or less continuous, although aggravated by violent exercise (jumping, etc.); or it may be that spoken of as renal colic—sudden severe cutting or stabbing pain in the region of one kidney radiating down the ureter to the bladder, testicle (which may become retracted), urethra, labia, or thigh; rarely to the opposite kidney or upward or about the chest. The severe pain is usually associated with strangury, reflex nausea, vomiting, and more or less shock (pallor, sweating, and feeble rapid pulse); and in children perhaps convulsions. The pain may last a few moments and then intermit or remit; or it may recur paroxysmally for several hours, the patient perhaps meanwhile being conscious of the transit of the calculus through the ureter. Finally, it may pass into the bladder and be discharged with the urine (with or without a painful paroxysm); or it may remain in the bladder and form the nucleus of a urinary calculus. Some dull aching usually remains for some time after the colic; and the colic may recur from time to time. Hematuria is very common; it occurs during and after an attack of colic, but it is usually present also in the absence of colic. Pyuria occurs in the event of pyelitis and pyelonephritis (which are common). There is also albuminuria (proportionate to the pus and blood), crystals of uric acid, calcium oxalate, etc. (that sometimes indicate the chemical constitution of the calculus); and sometimes also casts (concomitant nephritis). Anuria is sometimes observed; it may be due to bilateral calculi; to obstruction of one ureter and disease (atrophy) or reflex suppression of the function of the opposite kidney; or to urethral obstruction. In some cases, when the ureter is completely blocked and the opposite kidney healthy, the urine voided (excreted by the healthy kidney) may be entirely normal. Occasionally there is fever and other septicopyemic manifestations of pyelitis and pyelonephritis; in some cases there are recurrent attacks which recall Charcot's biliary intermittent fever (of cholelithiasis). The kidney may be tender to palpation, or enlarged (hydronephrosis, pyonephrosis). An impacted stone may be palpable along the course of the ureter (abdominal or vaginal or rectal palpation). An *x*-ray examination may reveal the presence, size, shape, and location of the stone or stones (uric-acid calculi give a faint shadow; oxalate and phosphate calculi, more marked shadows). Cystoscopic examination and examination of the separated urines may reveal whether one or both sides are diseased.

A calculus in the bladder gives rise to pain, especially at the end of urination, to sudden stoppage of the urinary stream, and to its re-establishment upon change of posture (from the erect to the recumbent); and to hematuria, pyuria, and other evidences of cystitis. It may be detected by bimanual and cystoscopic examination of the bladder.

Diagnosis.—The diagnosis of renal calculus is obvious in the event of renal colic, enlargement and tenderness of the kidney, hematuria, pyuria,

positive results of an *x*-ray examination, and the detection of gravel or sand; but a negative radiogram by no means excludes a stone. One must exclude other causes of pyelitis and pyelonephritis, renal tumors, tuberculosis of the urinary tract, essential renal hematuria; and renal colic must be distinguished from intestinal and gallstone colic, appendicitis, Dietl's crises, etc.

Prognosis.—The course is chronic and seldom (but sometimes) results in spontaneous cure after the passage of the then existing calculi. The likely complications (pyelitis, pyelonephritis) and the likelihood of recurrence render the prognosis doubtful.

Treatment.—Perhaps the dietetic regulations recommended in connection with gout may tend to prevent the formation of renal calculi. The treatment of renal colic is similar to that of gallstone colic—rest; morphine, atropine, and perhaps nitroglycerin hypodermicly; inhalation of chloroform, if necessary, until the morphine produces its effect; a hot foot bath, and hot applications locally. Inversion of the patient or the lateral posture sometimes serves to dislodge an impacted stone, and reverse massage may lead to the same result. Between attacks the patient should avoid sudden exertions. He should keep up a goodly excretion of urine by drinking large amounts of water; and in the event of uric acid calculi, he should reduce the urinary acidity with potassium citrate, acetate, or bicarbonate, etc. (or the alkaline mineral waters if desired); in the event of alkaline urine and phosphatic calculi, hexamethylenamin, salol, and other urinary antiseptics should be given. It seems quite impossible to dissolve calculi in the urinary tract, so that it is more than doubtful that lithium salts, piperazin, and other so-called uric-acid solvents have any such action in the body. In the event of repeated renal colic, pyelitis, anuria, etc., operation should be resorted to; and with a full knowledge of the condition, operation should not be too long delayed: sooner or later the renal tissue becomes destroyed and operation cannot restore it.

CYSTS OF THE KIDNEY.

Various sorts of cysts are encountered in the kidneys: (1) Small multiple cysts common in chronic indurative nephritis and attributed to obstruction and consequent dilatation of renal tubules or Bowman's capsules; (2) a large, often solitary cyst, sometimes found projecting from the cortex of an otherwise healthy kidney, containing clear watery or colloid fluid, and attributable also to obstruction of the tubules (perhaps a fœtal defect); (3) parasitic (echinococcic) cysts, which cause enlargement of the kidney, fluctuation, hematuria, pyuria, and, should the cyst rupture into the renal pelvis, vesicles, hooklets, or membrane in the urine (the passage of which may occasion renal colic); and (4) so-called congenital cystic kidneys.

Congenital cystic kidneys may be observed at birth (obstructing labor) or in later life; many cases first come to observation in adult

life, and especially in men. The kidney may attain an enormous size, being made up of numerous cysts, varying in size, as a rule, from 2 to 20 mm. in diameter, and containing urinous, colloid, or hemorrhagic fluid; often little if any renal tissue can be detected even microscopically. The condition is bilateral, although one kidney may be larger than the other, and perhaps enlarge before the other. The nature of the disorder is not definitely determined; it seems, however, to be a congenital defect of development (rather than retention cysts or cystic tumor formation, as has been suggested), since it is often associated with other anomalies of development), cystic disease of the liver, imperforate anus, malformation of the heart, etc.). The condition may remain latent until adult life, or, as seems to have occurred in some cases, slow progressive growth occurs from early life. In some cases there are the cardiovascular changes of chronic indurative nephritis.

Symptoms.—In some cases the enlarged kidneys interfere with parturition. In the cases that develop in later life there are: gradual enlargement of both kidneys (although one may precede), which may form visible and palpable (perhaps cystic) tumor masses; hematuria—chronic and remittent and induced by palpating the kidneys; and the gradual development of the urinary and cardiovascular changes of chronic indurative nephritis, and of chronic uremia; sometimes the sudden onset of acute uremia. Modern tests reveal lessening of the functional capacity of one or both kidneys.

Diagnosis.—The diagnosis can be readily made in the event of palpably enlarged kidneys, hematuria, and the urinary and cardiovascular changes of chronic indurative nephritis.

Prognosis.—The ultimate outlook is hopeless, although the subject may live many years (as in chronic indurative nephritis).

Treatment.—The treatment is that of chronic indurative nephritis. Operation may be undertaken in the rare unilateral cases in which the other kidney is of good functional capacity (modern tests), and in the event of suppuration; but the mortality is high and the outlook not reassuring.

TUMORS OF THE KIDNEY.

Tumors of the kidney may be benign or malignant, primary or secondary. The benign growths comprise fibroma, lipoma, angioma, and adenoma, and are of little clinical interest. The primary malignant growths comprise hypernephroma, sarcoma, and carcinoma. The hypernephroma, a tumor derived from misplaced adrenal tissue, is the most common tumor in adults, and includes most of the so-called adenomas, carcinomas, sarcomas, endotheliomas, and peritheliomas occurring in adult life. It may be small and an accidental finding at the necropsy, or it may attain the dimension of a man's head. In its early growth it is usually encapsulated against the renal substance; but later the capsule becomes invaded and penetrated, and the renal veins are

likely to be implicated—whence metastases occur by way of the blood-vessels, to the lungs, bones, etc. Sarcoma is the common tumor in infants and children, and may attain a very large size; it may be of the common types, but especially when occurring very early in life a rhabdomyoma, or a so-called congenital adenosarcoma, etc. Secondary tumors may occur by hematogenous or lymphogenous metastases or by growth by contiguity of tissue.

Symptoms.—The significant symptoms consist of pain, hematuria, a palpable tumor, and progressive anemia, cachexia, and emaciation. The pain may be slight, perhaps absent, or very severe; sometimes it is colicky (passage of blood clots down the ureter). Hematuria may be mild or severe, continuous or intermittent; it is present sometimes during the course of 50 to 90 per cent. of the cases, but in the early stages of hypernephromas it is usually wanting because of the encapsulation of the growth and its virtual separation from the kidney tissue. Sooner or later a tumor becomes palpable in the renal region retroperitoneally (behind the intestines and projecting into the flank or lumbar region); it varies in size, is soft or firm, and often nodular. In addition to blood, fluid or clotted (casts of the renal pelvis or ureter), the urine may contain bits of tumor tissue. Metastasis, especially in adults, to the lungs or bones may be observed in some cases; or growth of the tumor may lead to thrombosis of the inferior vena cava and œdema of the legs, varicocele (compression of the spermatic vein), or to compression and obstruction of the intestines, etc.

Diagnosis.—The diagnosis virtually is that of a retroperitoneal growth, in which event hematuria suggests its renal origin; progressive anemia and cachexia exclude other enlargements of the kidney (hydronephrosis, cystic disease), and the absence of fever and other evidences of septicopyemia excluded pyelonephrosis. Sarcoma of the retroperitoneum (Lobstein's cancer) must be excluded. Occasionally also tumors of the spleen or the liver must be differentiated.

Prognosis.—The prognosis depends entirely upon the possibility of removal of the growth.

Treatment.—The treatment is wholly surgical—removal of the tumor.

TUMORS OF THE BLADDER.

Tumors of the bladder comprise especially papilloma and carcinoma; fibroma and sarcoma are rare. The major *symptoms* of papilloma or carcinoma consist of local pain, disturbances in urination, hematuria, cystitis, and gradually developing anemia and cachexia. Sometimes the disorder is not suspected until a small bit of tumor tissue is discharged with the urine, whereupon microscopic examination reveals the true nature of the disease. In any case the *diagnosis* may be readily made by cystoscopic examination. The *treatment* is wholly surgical.

SECTION X.

DISEASES OF THE NERVOUS SYSTEM.

Pathological Physiology.—The nervous system, composed of two parts, the cerebrospinal and the sympathetic, is the chief regulating and governing mechanism of the organism. It is designed to exercise an orderly control over all the functions of the body—motor, sensory, secretory, vasomotor, and trophic; voluntary and involuntary; conscious and unconscious. The integrity of the nervous system is essential to the proper execution of purposeful acts; to muscular activity and coördination; to the reception of sensory impressions and the reaction thereto, as in reflex action, for instance; to the normal functioning of the special sense organs, and of the various glands; to proper growth and trophic influence; and to the normal activity of the cardiovascular, respiratory, digestive, urinary, cutaneous, and reproductive organs. Disorder of the nervous system thus becomes manifest not only in morbid phenomena referable directly to the system itself, but also in many varied disorders referable to the different systems of organs—which have already been discussed.

In the final analysis the nervous system is made up of a complex of neurons; that is, of certain ultimate anatomical and physiological units, each of which consists of a cell body, provided with a number of usually short, branching processes, so-called dendrons (dendrites), and a single, usually long, branch or process, the axon, axis cylinder, or nerve fiber. The axons of the cerebrospinal system soon after leaving the cell body become ensheathed with myelin and neurilemma (sheath of Schwann), both of which are lost near the termination of the axon; these medullary sheaths are absent in the sympathetic nervous system. The neurons are in physiological relationship or communication by means of so-called collateral branches or the terminal arborization of the axon of one neuron and the dendrons of another neuron. The prevailing conception is that there is no anatomical connection between physiologically related neurons, that the relationship is one of contact only—contiguity, but not continuity, of structure; but it is not impossible that the ultimate divisions of the terminal arborization of the axons actually enter the dendrons of another neuron (by means of so-called neurofibrils).

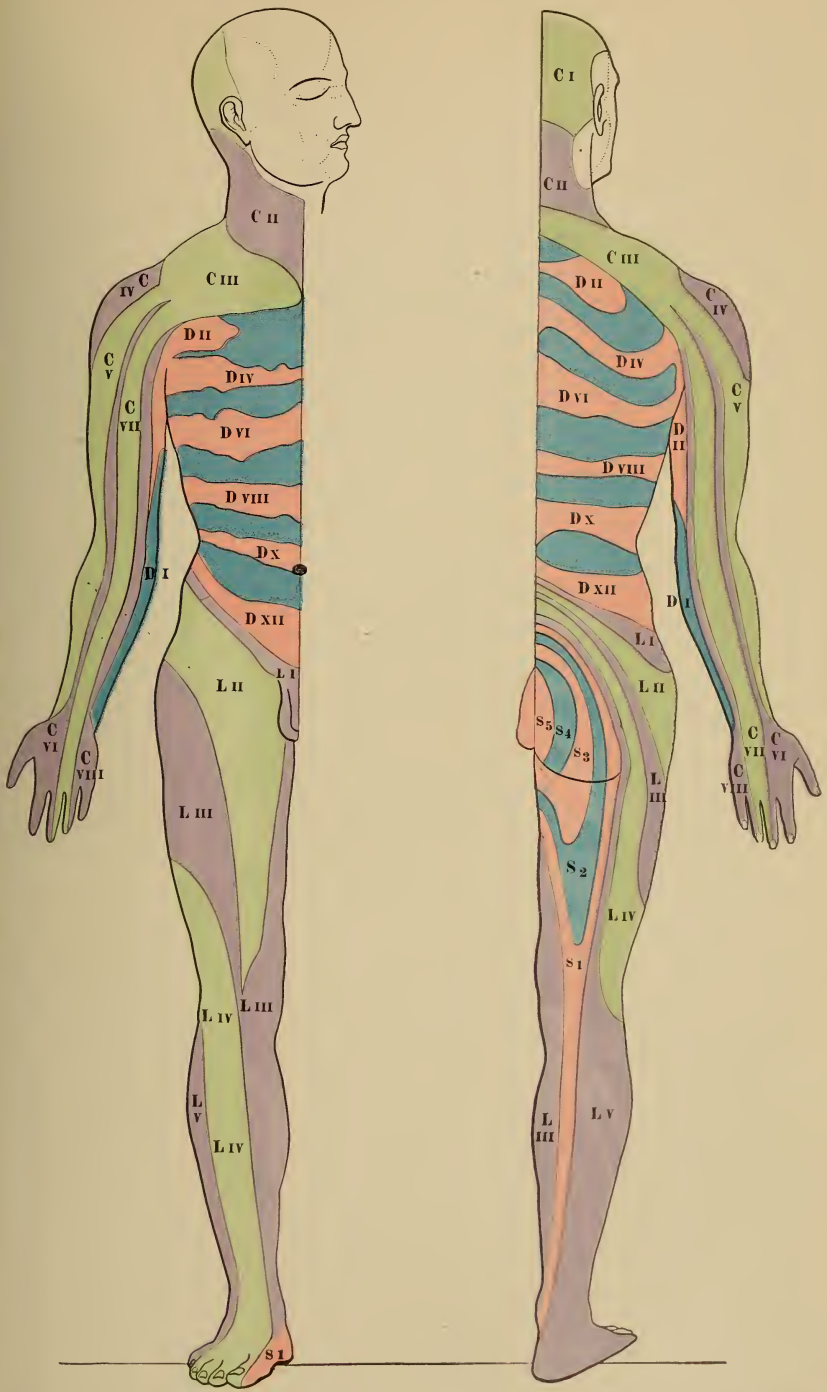
Neurons are classified as: (1) Primary neurons, which comprise those the cell body of which is situated in the central nervous system or ganglia,

and the axon of which extends to the periphery (skin, a muscle, a gland, a sense organ, etc.); and (2) secondary (or association) neurons, which comprise those the cell body of which is situated in the central nervous system and the axons of which do not extend to the periphery, but serve as a means of communication between different parts of the nervous system; that is, the axon communicates with the dendrons of another neuron (primary or secondary). Neurons also are classified, according to their function, into motor, sensory, secretory, and trophic; and a number of neurons, usually related functionally, are collected and held together by neuroglia and other connective tissue, and thus make up the different peripheral nerves, the nerve tracts of the spinal cord and brain, and the elements of the sympathetic system. Extensive communications exist between the cerebrospinal and the sympathetic systems—by way of the spinal roots and nerves with the spinal cord, and by way of the vagus (chiefly) with the brain. The sympathetic system is largely concerned with regulating the cardiovascular system and the vegetative functions; its activities are automatic and unconscious, although it is somewhat under the influence of the cerebrospinal system, and it may be deranged by emotional and other states; but we know comparatively little of the pathology of its disorders.

All disease of the nervous system is attributed to some disorder of the neuron. This may be supposedly functional in nature and temporary (functional disorders), or it may be organic, more or less destructive in nature, and susceptible of partial or complete repair (organic disease). The important facts are that when diseased the neuron is disordered (degenerates) as a whole; that many diseases of the nervous system tend to involve certain collections of neurons physiologically homologous (so-called system diseases); and that a knowledge of the anatomical and physiological relationships of these collections of neurons facilitates a comprehension of their diseases and permits a ready localization of the lesion.

Motor Neurons.—Motor impulses are conveyed from the cerebral cortex to the periphery by the motor neurons, of which there are two sets, the upper and the lower. The *upper motor neuron* (the motor tract) begins with the cell bodies in the motor area of the cerebral cortex; thence the axons pass through the corona radiata, the internal capsule, the crus, the pons, the medulla, and the spinal cord. In the pons and medulla most of the axons decussate, some to go to the nuclei of the motor cranial nerves, the others to course down the opposite side of the spinal cord, as the crossed pyramidal tract, and finally to break up into terminal arborizations about the cells of the anterior cornua (the cell bodies of the lower motor neuron); the axons that do not decussate in the medulla descend in the spinal cord as the direct pyramidal tract, but finally also decussate to the opposite side by way of the anterior commissure. The *lower motor neuron* begins with the cell bodies in the anterior cornua of the spinal cord; thence the axons, by way of the anterior (ventral) spinal roots become collected into the spinal nerves, and ultimately reach the muscles to which they transmit motor impulses.

PLATE I



Areas of Anæsthesia upon the Body after Lesions in the Various Segments of the Spinal Cord.

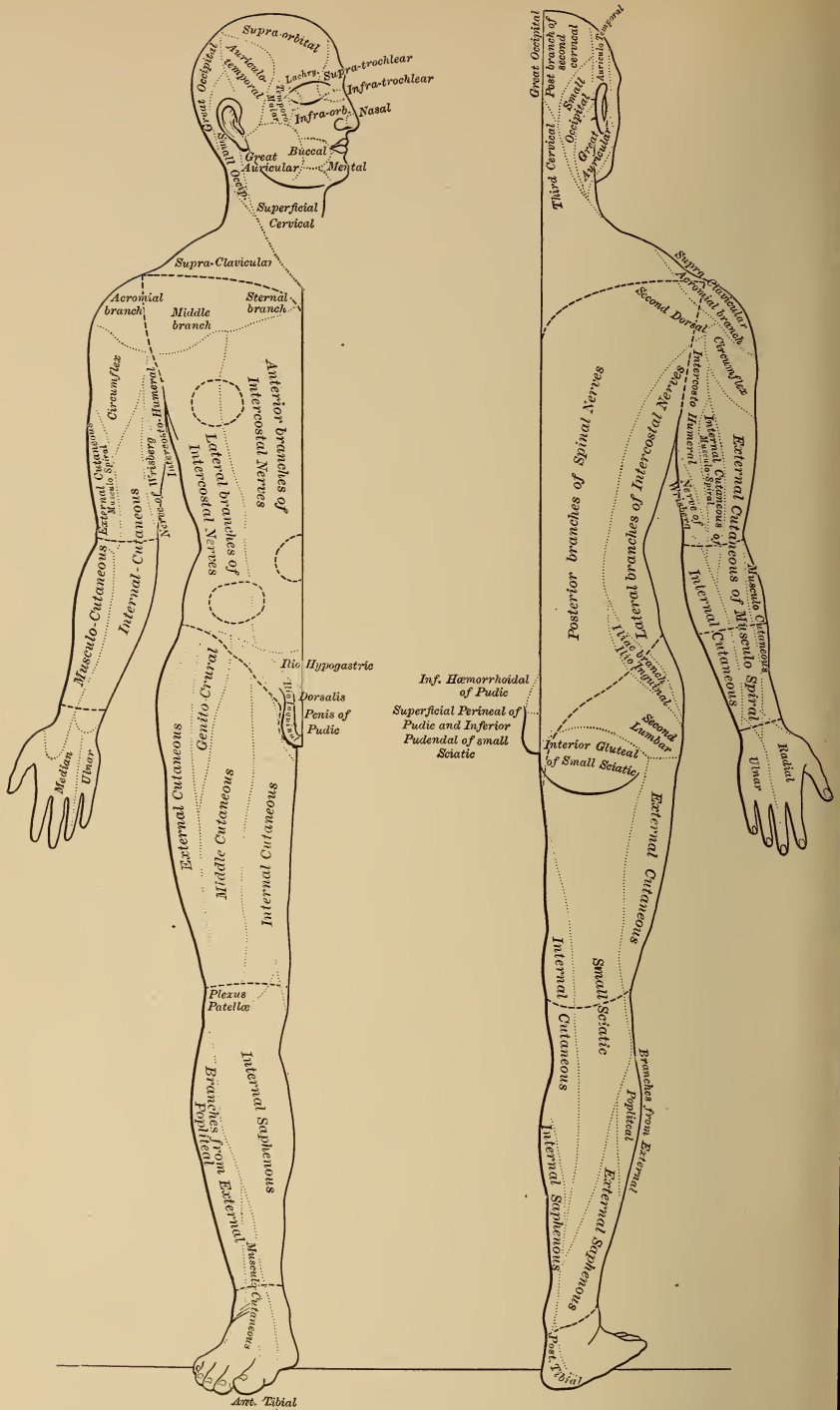
The segments of the cord are numbered: C I to VIII, D I to XII, L I to V, S 1 to 5, and these numbers are placed on the region of the skin supplied by the sensory nerves of the corresponding segment.

Motor impulses must pass through both of these neurons; and disease of one or the other or both results in loss of voluntary muscular power. There are, however, differences in the other results of disease of the two motor neurons, which are due to the fact that the upper neuron exercises an inhibiting influence on the lower, and that the lower serves to maintain the nutrition of the muscles with which it is connected. In general, therefore, disease of the upper motor neuron results in loss of voluntary muscular power, rigidity and spasticity of the muscles, and eventual contractures (the muscles of flexion and adduction overcoming those of extension and abduction); increase of the deep reflexes, as well as the Babinski plantar reflex (extension of the great toe); little if any muscular wasting, and that only from disuse; and no noteworthy changes in the electrical reactions of the nerves or muscles. This is well exemplified in lateral sclerosis (spastic paraplegia) and in combined lateral and posterior sclerosis (ataxic paraplegia). On the other hand, disease of the lower motor neuron results in loss of voluntary muscular power, flaccidity, and wasting of the muscles; ultimate contractures, due largely to the action of unopposed non-paralyzed muscles; loss of deep reflexes; and often the response to electrical stimulation known as the reactions of degeneration. This is well exemplified in epidemic poliomyelitis (infantile paralysis), progressive spinal muscular atrophy, progressive bulbar paralysis, etc. In some cases both motor neurons may be diseased, as in amyotrophic lateral sclerosis.

Sensory Neuron.—Sensory impressions are conveyed from the periphery to the nerve centres by the sensory neurons. The cell bodies of these are situated in the ganglia of the posterior spinal roots and in the homologous ganglia of the sensory cranial nerves, as well as in the sympathetic nervous system. The sensory impression is received by certain fine nerve filaments in the skin or tactile corpuscles and conveyed by a sensory nerve or bundle (through or past the cell body) to the spinal cord. Herein the axon bifurcates, one portion ascending in the posterior columns, the other descending a short distance.

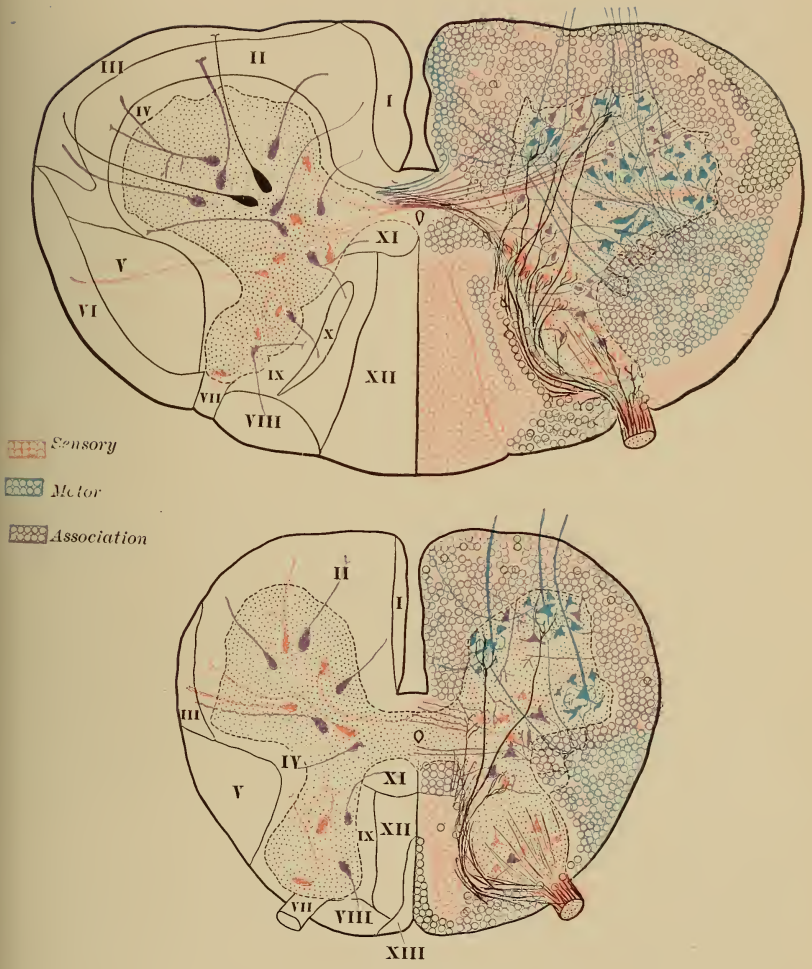
The peripheral nerves carry three systems of sensory fibers: (1) The protopathic system, which transmits sensations of pain and of extremes of temperature (above 45° C. and below 20° C.); (2) the epicritic system, which transmits sensations of light, touch, of cutaneous localization, and of slight temperature variations; and (3) the deep sensibility system, which transmits sensations of pressure and of movements of the joints, muscles, and tendons—so-called muscle sense (the ability to distinguish difference in weight of objects of similar size), as well as the stereognostic sense (the ability to recognize the shape of objects), and the ability to recognize with the eyes closed the position into which the limbs may be passively placed. The unit of the protopathic system is in the posterior spinal roots, that of the epicritic system in the peripheral nerves; so that the areas of sensory disturbances are different in disease of the peripheral nerves (Fig. 15) from what they are in disease of the spinal roots or segments (Plate I). Furthermore, after injury to a peripheral nerve, for

FIG. 15



The distribution of the sensory nerves in the skin. The areas of the skin supplied by the cutaneous nerves are shown in finely dotted outline. The circles on the trunk show areas occasionally anesthetic in hysteria. The lines across the limbs at the ankle, knee, thigh, wrist, elbow, and shoulder show the upper limits of anesthesia in multiple neuritis of varying degrees of severity. (After Flower.)

PLATE II



The Cervical and Sacral Enlargements of the Spinal Cord in Cross-section. (Starr.)

Showing the various neurons in the gray matter, the direction of their axons, and the varieties of fibers in the different columns of the cord. Blue, motor neurons; red, sensory neurons; purple. association neurons and axons.

I. Ant. median column. II. Anterolateral column. III. Gowers' anterolateral ascending column. IV. Marginal column. V. Lateral pyramidal column. VI. Direct cerebellar column. VII. Lissauer's tract. VIII. Ext. portion of column of Burdach. IX. Root zone of the column of Burdach. X. Descending comma-shaped bundle of Schultze. XI. Post. commissural tract. XII. Column of Goll. XIII. Septomarginal tract.

instance, the different forms of anesthesia are not alike in distribution, and during recovery the sensibility to pain returns before that of mere touch; that is, sensation for touch and pain is subserved by different fibers. If a peripheral nerve represents largely a single posterior spinal root, the area of analgesia is likely to be well defined; but if the peripheral nerve contains fibers from several posterior spinal roots, the disturbances of protopathic sensibility are likely not to be well demarcated. After all the cutaneous sensory fibers have been destroyed, the deep sensibility of the part may still be preserved.

Sensory impressions are transmitted in the spinal cord by the following tracts (Plate II): (1) The posterior columns—the postero-internal column of Goll, and the postero-external column of Burdach. The fibers of these columns are a continuation of those of the posterior roots; they ascend, giving off collateral branches which penetrate the gray matter of the cord, and they terminate in the homolateral nucleus gracilis and nucleus cuneatus of the medulla; thence another set of fibers proceeds, soon decussates, and ends in the thalamus of the opposite side; from this, in turn, another set of fibers proceeds to the sensory area of the cerebral cortex. These fibers are chiefly concerned in transmitting impulses of cutaneous tactile sensibility (light touch) and localization, and of deep sensibility, especially muscular and limb position; lesions of these fibers give rise to ataxia. A few fibers cross soon after they enter the cord, ascend in contralateral ascending anterior column, and finally terminate in the thalamus. These fibers also transmit impulses for tactile sensibility, and may escape involvement in cases of Brown-Séquard paralysis. The posterior columns also contain fibers concerned in the transmission of painful impressions; soon after entering the cord they terminate about cells in the gray matter, whence other axons proceed, decussate, and ascend in the opposite anterolateral tract. (2) The anterolateral tract of Gowers. This comprises the aforementioned fibers for pain, as well as others concerned in transmitting temperature impulses; these ultimately, in large part, reach the cerebellum, by way of the superior pedicle), but some of them proceed to the homolateral thalamus. (3) The direct (dorsolateral) cerebellar tract of Flechsig. Some of the fibers entering the cord by way of the posterior roots, proceed to cells in Clark's columns (at the base of the posterior cornua); thence axons proceed, ascend as the direct cerebellar tract, and end in the cerebellum. These fibers convey impulses of deep sensibility (muscles, joints, tendons, etc.), and disorder of them results in ataxia.

Reflexes.—The contraction of a muscle or set of muscles that follows a sensory impulse is known as a reflex act; its occurrence is dependent upon the integrity of the so-called reflex arc. This is composed of (*a*) an afferent (sensory) nerve, along which the impulse passes, enters the spinal cord by way of the posterior roots, and reaches (*b*) the so-called centre, the motor cells of the anterior cornua; from these, in response to the stimulus, an impulse passes out along (*c*) an efferent (motor) nerve, and, reaching a muscle, causes it to contract. Reflexes

may occur in the event of integrity of a single segment of the spinal cord; but, although independent of, they are much modified by, the higher centres in the brain. In disease they may be lessened (or abolished) or exaggerated. Lessening or absence of the reflexes implies a lesion in either the sensory nerve, in which event it is associated with anesthesia; or in the motor cells in the anterior cornua, or in the motor nerve, in which event it is associated with paralysis, flaccidity, and wasting of the muscles; or in the sensory reflex fibers within the spinal cord, in which event the reflexes being diminished or absent, without associated sensory (anesthetic) or motor (paralytic) phenomena referable to a nerve trunk, means a lesion of the spinal cord. Of these, the most important are locomotor ataxia, paretic dementia (with associated posterior sclerosis), acute and chronic anterior poliomyelitis, syringomyelia, diffuse and transverse myelitis, as well as hemorrhage and tumors (which abolish reflexes at the level of the lesion), and multiple sclerosis (which also may abolish a reflex if a sclerotic patch involves the reflex arc). Exaggeration of the reflexes occurs in consequence of lessening or abolition of the inhibiting influence of impulses normally passing from the brain by way of the motor tracts in the lateral columns of the spinal cord; the exaggeration may progress to the condition known as clonus, which comprises a series of rapidly recurring contractions of a muscle, provoked by sudden marked tension on the tendon of the muscle. Exaggerated reflexes are indicative of disease of the lateral columns of the spinal cord, and are found especially in lateral sclerosis (spastic paraplegia), below the lesion in cases of descending degeneration of the pyramidal tracts, in the early stages of amyotrophic lateral sclerosis (later they become lost in the arms), in conditions of slight pressure impeding the passage of inhibiting impulses down the cord (as in the early stage of caries of the vertebræ, tumors, etc., causing slight compression), and in functional disorders, such as hysteria and neurasthenia. In cases of complete severance of the cord, however, the reflexes are abolished below the lesion.

There are three principal classes of reflexes: (1) The deep or tendon reflexes, which result from tapping a tendon near its insertion, whence contraction of the muscle ensues. The most important are the knee-jerk (patella tendon reflex), the Achilles tendon reflex, the biceps, the triceps, the supinator longus, the plantar (flexion) tendon reflex, etc. Special diagnostic importance attaches to the knee-jerk: it may be diminished or lost in disturbance of the reflex arc interfering with the transmission of impulses, such as disease of the muscles, nerves (neuritis), posterior roots (tabes dorsalis), or anterior roots, and disorders of the lumbar cord (second, third, and fourth lumbar roots) which destroy the centre (acute and chronic poliomyelitis, etc.). In some cases, occasionally, but only for a very short time, the knee-jerks may also be lost—from irritation or stimulation of the inhibiting influence of the pyramidal fibers—in cerebral hemorrhage, meningitis, compression of the cord from fracture-dislocation, etc. The knee-jerks may be exaggerated in consequence of lessening or abolition of the inhibiting influence of the pyram-

idal tract above the lumbar region, such as occurs in lateral sclerosis (spastic paraplegia), transverse myelitis, compression from fracture-dislocation or other causes (except in cases of complete severance of the cord, in which event the reflexes below the lesion are abolished); exaggeration of the knee-jerk occurs also in functional disorders, such as hysteria and neurasthenia. Under like conditions, exaggeration of the Achilles tendon reflex—that is, ankle clonus—may ensue, and the so-called Babinski phenomenon (or sign) may be elicited; this, which consists of extension (sometimes called dorsal flexion) of the great toe upon stimulating the sole of the foot, is proof of disease of the upper motor neuron. (2) The superficial or cutaneous reflexes, which are elicited by stroking or irritating the skin. The most important are the epigastric reflex—contraction of the muscles of the epigastrium upon stroking the lower part of the thorax; the cremasteric reflex—retraction of the testicle and scrotum upon stroking the inner aspect of the thigh; the gluteal reflex—contraction of the gluteus muscle (dimpling in the gluteal fold) on stroking the buttock; and the abdominal, scapular, palatal, pharyngeal, and conjunctival reflexes. Most of these reflexes are believed to be due to contraction of involuntary muscular fibers beneath the skin near the point of irritation. The presence of these superficial reflexes implies integrity of the reflex arc, whence they may serve to determine the level of disease of the spinal cord. In general they are diminished or absent in diseases in which the deep or tendon reflexes are exaggerated. (3) Visceral reflexes, of which the most important are: (*a*) pharyngeal and laryngeal reflexes, which, when lessened or abolished, result in difficulty in swallowing and coughing, and may lead to regurgitation of food through the nose, or to aspiration pneumonia or asphyxiation; (*b*) visceral and rectal reflexes. Disease of the spinal cord above the centre for the bladder and the rectum (fourth and fifth sacral segments) results in loss of control by higher centres, so that the bladder must depend upon its reflex activity; usually it becomes overdistended and finally overflow from distention ensues. If the spinal centres are destroyed retention of urine is usually impossible, and there is continuous incontinence. The result of involvement of the rectal centre is constipation and involuntary evacuation of the feces.

Electrical Reactions.—Normally the application of a faradic (interrupted) or galvanic (constant) current to a nerve or muscle results in contraction of the muscle, especially when the current is applied to the so-called motor points. On the use of the galvanic current, contractions occur only when the contact is made or broken; and normally the cathodal (negative pole) closing contraction (CCC) is greater than the anodal (positive pole) closing contraction (ACC). In lesions of the lower motor neuron, usually within four or five days, the so-called reactions of degeneration develop. The nerve shows a gradual loss of excitability to both the faradic and the galvanic currents, and finally fails to respond at all, that is, there is no muscular contraction upon applying the current to the nerve. The muscle at first responds feebly and slowly to the

faradic current, but soon ceases to respond; on the other hand, the muscle may be for some time unduly sensitive to the galvanic current, that is, it may respond to a weaker current than in health; but the characteristic reactions of degeneration consist in a slow, wave-like contraction to the galvanic current (instead of instant and quick response, as in health), and in the anode-closing contraction being greater than the cathode-closing contraction. This last is sometimes not present (partial reaction of degeneration). In severe cases all response to even the galvanic current may become lost. A gradual increase in galvanic irritability in cases in which it has been absent or slight is of good prognostic significance, indicating regeneration.

DISEASES OF THE NERVES.

NEURALGIA.

Neuralgia is a term used to describe recurring or persistent pain in a peripheral nerve and its area of distribution. Strictly speaking, the pain is unassociated with demonstrable lesions in the nerve trunk or its centres, but the pain of definite neuritis is also often spoken of as neuralgia.

Etiology.—Neuralgia occurs especially in neuropathic individuals or families, and is more common in women than in men. It is commonly provoked by anemia and general nervous debility; but it may follow "taking cold," or be due to infections, such as malaria, typhoid fever, rheumatism, etc.; to intoxications, such as intestinal auto-intoxication, Bright's disease, gout, etc.; or to reflex irritation, such as carious teeth, eyestrain, etc.

Pathology.—Neuralgia *per se* is not associated with demonstrable anatomical lesions. The pain is very likely due to irritation or oversusceptibility of the sensory neuron.

Symptoms.—The chief symptom is pain. Although this may be more or less constant, it is characteristically intermittent, recurring from time to time (seconds, minutes, or longer intervals) in paroxysms of sharp, stabbing, lightning-like aching or boring pain, which is limited to the area of distribution of a nerve or group of nerves. Usually there is also hyperesthesia over this area, and painful or tender spots along the course of the nerve; and there may be vasomotor alterations, such as pallor or flushing, excessive sweating, erythema, or œdema, and trophic changes, such as blanching and falling of the hair. Reflex spasms of the muscles in the painful area sometimes supervene, as in trigeminal neuralgia. The attack may last several minutes, several hours, or a day or more, and recur at regular or irregular intervals.

Trigeminal neuralgia (*trifacial neuralgia; tic douloureux*), a very common disorder, may be symptomatic of carious teeth, eyestrain, nasal

disease, brain tumor, etc., or it may occur as an apparently primary disorder of unknown cause. The pain, which is often extremely severe, may be limited to any one of the three great divisions of the nerve (the ophthalmic, the superior and the inferior maxillary), but it may involve all three; it is rarely, if ever, bilateral. Cutaneous hyperesthesia is usually present, painful or tender spots are commonly found where the nerve emerges from the foramina, and the eyebrows and the hair are likely to become gray or fall out. The paroxysms may be set up by various sorts of stimuli, such as a draught of cold air, touching the hyperesthetic area, the movements of chewing, speaking, etc.; they tend to increase in severity and the intervals to lessen; and associated spasm of the facial muscles may supervene (facial spasm).

Cervicoöccipital neuralgia occurs in the back of the head, in the area of distribution of the first four cervical nerves; it may be confined to the posterior branches. There is often a tender spot midway between the mastoid process and the first cervical vertebra where the great occipital nerves emerge, between the trapezius and the sternomastoid muscle, and above the parietal eminence. The scalp is usually markedly hyperesthetic.

Cervicobrachial neuralgia occurs in the shoulder and arm, in the area of distribution of the four lowest cervical and the first thoracic nerves (brachial plexus). Tender spots may be found at the posterior border of the deltoid muscle (circumflex nerve), over the musculospiral nerve near the outer condyle of the humerus, over the ulnar nerve in the groove between the inner condyle and the olecranon process, and over the ulnar and the radial nerves at the wrist.

Phrenic neuralgia sometimes follows pleurisy or pericarditis, and only very rarely occurs as a primary disorder. The pain occurs at the lower part of the chest, in the neighborhood of the attachment of the diaphragm, and is aggravated by deep inspiration, coughing, sneezing, and other sudden or violent movements of the diaphragm.

Intercostal neuralgia is very common. The pain follows the course of the intercostal nerves, and is often continuous, though subject to acute exacerbations. Painful spots are common near the vertebræ, where the posterior branches emerge; in the midaxilla, where the lateral branches reach the surface; and toward the anterior midline, where the anterior branches reach the surface. The most intractable intercostal neuralgia (as well as other neuralgias) may follow herpes zoster.

Lumbar neuralgia occurs in the area of distribution of the sensory fibers of the sacral plexus, and is most common along the crest of the ilium, in the inguinal region, and in the genitalia (scrotum and labia).

Coccygodynia, a neuralgia of the coccygeal plexus, is common in women. The pain may be more or less constant and intractable, and is usually aggravated, or at least induced, by the sitting posture.

Sciatica may be a neuralgia, although it may result also from neuritis, pressure of tumors, enlarged lymph nodes, sacro-iliac disease, etc. The pain, which is often severe, may extend throughout the course of the

nerve and into its branches in the leg. Hyperesthesia is usually present, and painful spots may be found at the sciatic notch, in the popliteal space, and at the outer edge of the fibula. There is usually pain and difficulty in walking, and the common attitude in standing and walking (throwing the weight on the unaffected side) may lead to lateral curvature of the spine.

Meralgia paræsthetica is a neuralgia (or neuritis) of the external cutaneous nerve that gives rise to painful paresthetic phenomena on the outer side of the thigh, and sometimes also some anesthesia.

Plantar neuralgia may occur in the tips of the toes, or it may be more widespread. It is rather common in typhoid fever.

Painful heel (pododynia) may be a pure neuralgia, but in some cases it results from definite exostoses and infiltrations of the plantar fascia. They are not uncommon after gonococcic infection, and may be demonstrated by the *x*-rays. Pains in the feet also are often due to flat foot.

Metatarsalgia, Morton's painful affection of the foot, is usually attributed to compression of the metatarsal nerve (usually about the fourth metatarsal phalangeal joint).

Visceral neuralgias are sometimes described, such as cardialgia, gastralgia, hepatalgia, nephralgia, etc., but these painful affections usually have an anatomical basis.

Diagnosis.—Neuralgia should be distinguished from definite neuritis. In neuralgia there is no discoverable cause for the pain; the pain is usually unilateral, corresponds with the area of distribution of a nerve, and is characteristically paroxysmal and intermittent; there is no noteworthy anesthesia (rather hyperesthesia), no muscular wasting, and no reactions of degeneration; in neuritis there is early and usually marked tenderness along the nerve trunks. The pains of tabes dorsalis, of gummas, and of inflammation of the meninges should not be mistaken for neuralgia.

Prognosis.—The neuralgias of general ill health and anemic states usually disappear with the causative factors. Trigeminal neuralgia, sciatica, and the pain following herpes zoster, etc., may continue for a long time—sometimes indefinitely.

Treatment.—Possible causative factors must be searched for, such as carious teeth, refractive errors, nasal disease, pressure of tumors, anemia, nervous exhaustion, gout, etc., and, when found, these must be appropriately treated. Iron and arsenic are useful in any event; in some cases also phosphorus, cod-liver oil, and strychnine. Quinine is of value in cases definitely malarial, and perhaps in some other cases, for its bitter tonic properties. The iodides sometimes render service in chronic cases and in those associated with arteriosclerosis, gout, etc. Alkalies, such as sodium benzoate, ammonium chloride, etc., gelsemium, and cannabis indica also have been recommended, as well as injections of alcohol into the nerve. Otherwise, the treatment is largely that of pain: hot applications, freezing the skin (ether or rigoline spray), the Paquelin cautery, blistering, the application of equal parts of camphor, chloral, and menthol, and galvanism locally; and the internal use of the coal-tar analgesics (acetphenetidin, acetanilide, etc.). These, as well as preparations of

opium, should be used with the greatest caution, since the common intractability of neuralgia is likely to lead to the formation of a drug habit. In many cases surgical treatment must eventually be resorted to—resection of the nerve (or of the Gasserian ganglion in trigeminal neuralgia).

NEURITIS.

Etiology.—Neuritis may be acute or chronic, and it may involve one or many nerves—mononeuritis or multiple neuritis. Single or mononeuritis may be due to traumatism, to the action of cold, or to extension of inflammation from adjacent parts, as facial neuritis in middle-ear disease and caries of the temporal bone. Multiple neuritis may be due to infections (diphtheria, typhoid fever, pyococcic infections, leprosy, etc.), and those factors associated with "catching cold" and exposure (acute febrile polyneuritis); exogenous intoxications, such as alcohol, lead, arsenic, and mercury, and that associated with beri-beri; endogenous intoxications associated with gout, diabetes, nephritis, intestinal auto-intoxications, etc.; anemic and cachectic states.

Pathology.—In general the lesions are those of inflammation (congestion, œdema, and cellular infiltration), fragmentation of the myelin sheaths, and parenchymatous swelling and ultimate fatty and granular degeneration of the nerve fibers. The lesions may be superficial, chiefly perineural, or they may penetrate to the deeper parts—so-called interstitial neuritis. In some cases (so-called parenchymatous neuritis) the axons are chiefly involved, showing degenerative changes alike to those that follow isolation of the axon from the cell body. The cell bodies also may reveal changes showing that the etiological factor tends to involve the entire lower motor neuron.

Symptoms.—Since most of the peripheral nerves carry motor, sensory, and trophic fibers, the symptoms of neuritis may be also motor, sensory, and trophic. In *single* or *mononeuritis* the chief complaint is usually of pain in the course of the nerve and in its area of distribution. In addition there is usually hyperesthesia and perhaps hyperalgesia or paresthesia of the overlying skin; in some cases also patches of hypesthesia or anesthesia, or sometimes erythema or œdema. Furthermore, there is loss of power of the muscles, which soon become flabby, wasted, and tender (especially on motion or pressure), and reveal fibrillary twitchings and the reactions of degeneration. The reflexes become lessened and may disappear. The involved nerve, if accessible to palpation, is found swollen and tender. In protracted cases, in addition to the foregoing, more or less marked trophic changes occur: the skin becomes thin, smooth, red, and shiny or glossy; the tips of the fingers become pointed (from atrophy also of the subcutaneous tissues); the nails and the hair grow irregularly; and the joints may become stiff and immobile.

In *multiple neuritis*, since the poison is carried by the blood, the lesions are widespread and usually symmetrically bilateral. In the acute

febrile (infectious or toxic?) cases the onset, following cold or exposure, may be sudden, and attended by a chill or chilliness, fever (103° to 104°), pains and tenderness of the limbs, and loss of muscular power. The other symptoms mentioned under mononeuritis develop, but are widespread, and the patient is usually totally incapacitated.

The *alcoholic* (the most common) cases usually begin with paresthesia (tingling or numbness) and hyperesthesia, especially in the legs, and these may be associated with tenderness of the calf muscles. Soon weakness of the muscles of the legs, especially of the anterior tibial group, develops; this leads to "foot-drop" and to a characteristic high-steppage gait. There is also atrophy of the muscles, loss of the knee-jerk and the plantar reflex, the reactions of degeneration, incoördination (loss of muscle sense), and other phenomena mentioned under mononeuritis. In advanced cases walking finally becomes impossible (complete paralysis), and areas of anesthesia (stocking-and-glove, or cuff forms) supplant the hyperesthesia. The arms may be involved, but less commonly and less severely. Although the cranial nerves usually escape, vision may become impaired, optic neuritis may occur, and involvement of the phrenic nerve (paralysis of the diaphragm) or of the vagus nerve (tachycardia) may ensue. Mental phenomena are not uncommon in aggravated cases, such as loss of memory, hallucinations, mental deterioration, and delirium. Some chronic alcoholic subjects exhibit the combination of multiple peripheral neuritis, loss of memory, and pseudoreminiscences (false notions as to time, place, and actualities)—a condition spoken of as Korsakoff's psychosis.

The *arsenical* cases much resemble the alcoholic; the legs are usually involved first, but the arms may become implicated later, and there is usually also a brownish pigmentation of the skin. In the cases due to lead the extensors of the wrist and hand are most likely to become involved (wrist-drop), and the other symptoms mentioned on page 295 develop. In the *diphtheritic* cases the muscles of the palate are commonly involved and lead to regurgitation of food through the nose; but the external muscles of the eye may become affected or the disorder may become widespread.

Diagnosis.—The diagnosis is usually apparent from the mentioned symptoms. The neural distribution of the motor and sensory symptoms, tenderness of the nerve trunks and the muscles, the bilateral symmetry of the phenomena in the multiple neuritis cases, and the absence of involvement of the sphincters of the bladder and the rectum and of the Argyll Robertson pupil usually serve to exclude spinal-cord disease.

Prognosis.—The outlook in general is good, gradual improvement occurring in most cases after the lapse of several weeks or months. Recovery, however, may not be complete, and death may ensue from complications, such as bronchopneumonia.

Treatment.—The causative factor must first be entirely eliminated—especially alcohol, arsenic, lead, gastro-intestinal autotoxemia, etc. Rest and good nutritious food are essential in all cases, and care must be

exercised that deformities do not ensue. The initial pains may be relieved by local applications, such as hot fomentations, saturated solutions of magnesium sulphate, liniments containing chloroform, belladonna, etc., and by the internal use of salicyl preparations or small doses of acetphenatidin and other coal-tar derivatives. After the acute manifestations have subsided, resort should be had to massage, passive movements, and galvanism to maintain the nutrition of the muscles, and the patient should be encouraged to attempt voluntary movements. Strychnine in large doses is useful in many cases, and iron, quinine, cod-liver oil, and other tonics in the anemic cases.

HERPES ZOSTER.

Herpes zoster is an acute, probably specific, infectious inflammation and degeneration of the ganglia of the posterior spinal roots and of the homologous ganglia of the sensory cranial nerves, characterized clinically by fever, and by pain, erythema, and an herpetic eruption in the area of distribution of one or more of these roots.

Etiology.—Head and Campbell seem to have proved that herpes zoster is an acute specific infectious disease, involving especially the posterior spinal roots and ganglia, but the infectious agent has not been determined. The disorder also sometimes develops secondarily to tuberculosis of the vertebræ, meningitis, etc.

Pathology.—The chief lesions consist of inflammatory foci, with necroses and hemorrhages involving the affected ganglion and its sheath. Many ganglion cells are destroyed, and in consequence their axons and dendrons in the peripheral nerves and the spinal cord undergo secondary atrophy. The disease in many respects is the sensory analogue of acute poliomyelitis. The facial herpes of pneumonia and other infections is an analogous condition (involvement of the Gasserian ganglion).

Symptoms.—The onset may be abrupt, with fever, malaise, gastrointestinal disturbances, perhaps enlargement of the lymph nodes, etc. Usually on the third or fourth day, and often preceded by pain and tenderness of the skin, an herpetic (or vesicular) eruption breaks out. This is most common on the trunk (intercostal nerves) and is usually unilateral; it is confined to the area of distribution of one or more posterior spinal roots (segmental distribution), and it is especially characterized by a patchy distribution of the vesicles corresponding to the posterior, lateral, and anterior division or distribution of the nerves. At the end of a week or ten days the vesicles usually dry up. Small depressed scars commonly persist; sometimes also a postherpetic pain and various sensory disturbances, and occasionally a most intractable neuralgia. Transient motor paralysis may be observed, especially of the muscles of the facial nerve innervation in trigeminal or occipito-cervical nerve herpes (compression of the facial nerve by a swollen geniculate ganglion, Ramsey Hunt).

Treatment.—The early pain may be relieved by coal-tar derivatives, salicyl preparation, or opium. Zinc stearate or similar dusting powder, or a mild antiseptic ointment may be applied locally. Intractable neuralgia, if not otherwise relieved, may necessitate division of the posterior spinal roots involved or removal or isolation of the homologous cranial nerve ganglion.

NEUROMAS.

Neuromas may be true or false. The true neuroma is a tumor made up of nerve fibers, and sometimes also of ganglion cells; it is a rare growth. The false neuromas are of divers sorts: (1) Various forms of fibromas, myxomas, or sarcomas that may develop from the connective tissues of the nerves, and usually give rise to fusiform swellings. (2) The so-called *amputation neuromas*—painful, bulbous enlargements of the ends of divided nerves. They are common in stumps after amputations, and consist of a mass of axons growing peripherally—supposedly with a view to reach their normal endings. (3) *Plexiform neuromas*—a condition, often hereditary and congenital, consisting of multiple fibrous tissue growths that may involve almost if not quite all the nerves of the body. More than a thousand of these tumors have been found in a single case. (4) *Generalized neurofibromatosis* (*von Recklinghausen's disease*), a condition consisting of: tumors along the nerves akin to those of the plexiform neuroma; various motor and sensory phenomena depending upon the situation of these growths; distorted mentality, consisting especially of general failure of intellectuality and disorders of speech; soft, sessile, or pedunculated growths of the skin (neurofibromatosis of the skin; molluscum fibrosum; so-called elephantiasis neuromatosa, when of great size); and areas of brownish pigmentation of the skin. (5) *Tubercula dolorosa*, small, painful, multiple neuromas involving the cutaneous terminations of the sensory nerves, and found especially in the breast, on the face, or about the joints. (6) *Dejerine's interstitial hypertrophic progressive neuritis of childhood* (thickening of the nerves, with general muscular atrophy and the symptoms of tabes dorsalis; page 856).

DISEASES OF THE CRANIAL NERVES.

I. **Olfactory Nerve.**—The olfactory nerve may become diseased in the terminal cells, in the olfactory mucous membrane, in the cells in the olfactory bulb, in the olfactory tracts, or in the cortical cells in the uncinate convolution. The disorders consist of: (1) Anosmia or hypoösmia (loss of the sense of smell), which may occur in chronic nasal catarrh, disease of the bulb or tracts (injuries, meningitis, etc.), central disturbance, or hysteria (usually unilateral); (2) hyperosmia, which is commonly observed in hysterical and neurasthenic women; and (3) parosmia—perversion or hallucinations of smell—which may occur after

injuries, in tumors of the uncinate convolution, in the insane, and may constitute the aura of epilepsy.

II. **Optic Nerve.**—**Functional Disorders.**—Functional disorders of the retina are sometimes observed, such as hyperesthesia, which is occasionally seen in hysterical women and in the early stages of meningitis (photophobia); night blindness—nyctalopia, good vision in a strong light and poor vision in twilight; and hemeralopia, good vision in twilight and poor or painful vision in a bright light; hysterical amblyopia or amaurosis, which is encountered in hysterical subjects; and toxic amblyopia or amaurosis, which may be due to tobacco, lead, quinine, etc. In the toxic, especially tobacco, cases there is gradual bilateral impairment of sight, especially centrally and for colors (central scotoma for red and green). In advanced cases a retrobulbar neuritis may ensue, with atrophy of the macular bundle and of the disk.

Neuroretinitis.—Neuroretinitis is common in chronic nephritis, severe anemic conditions, and leukemia, and it may be observed also in syphilis, malaria, diabetes, lead poisoning, etc. The chief lesions consist of hemorrhages and opacities of the retina. The disorder may be well advanced before there is any notable impairment of vision.

Optic Neuritis; Papillitis; Papillœdema; Choked Disk.—Papillœdema is sometimes seen in chronic nephritis, severe anemic states, etc., in which it is usually associated with neuroretinitis; but it is especially significant of increased intracranial pressure, and is therefore most common in brain tumors (at least 90 per cent. of the cases), brain abscess, and meningitis (in which it may be due to extension of the inflammation). The lesions are usually bilateral, and consist of congestion and swelling of the disk, blurring of its edges, and filling of the physiological cup. Often there is an associated neuroretinitis. The process may subside, but optic atrophy is the common sequel. The early stages of the disorder are to be recognized only by ophthalmoscopic examination, since considerable progress may be made before there is notable impairment of vision.

Optic Atrophy.—Optic atrophy may occur secondarily to the causes that set up papillitis. Primarily it occurs as (1) a congenital, family disease (Leber's disease), most common in adolescent males, but usually transmitted by the females; and (2) in tabes dorsalis, paretic dementia, and sometimes in other nervous disorders. The lesions consist of gradually increasing pallor of the disk, with a well-defined edge, which in the secondary form may be irregular in outline; the artery may appear normal or become small. The symptom is gradual failure of vision.

The Optic Chiasm.—The chiasm is most frequently disordered by tumors of the brain, especially of the pituitary body (akromegaly). The fibers that supply the inner half of each retina (temporal field of vision) are commonly first involved, so that the symptom is double temporal hemianopia. Eventually, with progress of the disease, total blindness may ensue. Rarely two symmetrically situated tumors may implicate the fibers proceeding to the outer half of each retina (nasal field of vision) and bilateral nasal hemianopia may ensue.

The Optic Tract and Centre.—Disease of the optic tract between the chiasm and the cortex (cuneate portion of the occipital lobe) results in failure of vision of the temporal half of the retina of the corresponding side and of the nasal half of the retina of the other side (partial or complete homonymous hemianopia). The lesion may be in the fore part of the optic tract, in or about the thalamus, the lateral geniculate body, the corpora quadrigemina, the posterior part of the internal capsule, the optic radiation, or the cuneus. Disease of the supramarginal and angular gyrus may result in crossed amblyopia (concentric diminution of both fields of vision, usually more marked on the side opposite the lesion) and visual aphasia (word blindness, mind blindness). Resort to Wernicke's hemiopic pupillary reaction may enable one to determine whether the lesion is in advance of or posterior to the corpora quadrigemina: if throwing a bright light into the blind half of the retina results in contraction of the pupil, it shows integrity of the reflex arc (the optic nerve fibers, the centre for the pupil, and the third nerve), and the lesion is therefore in the optic radiation or the visual centre in the cuneus. Hemianopia usually means an organic lesion; rarely it may be found in hysterical disorders. Various sorts of aphasia may be associated with it.

III. **Oculomotor Nerve;** IV. **Trochlear Nerve;** VI. **Abducens Nerve.**—The third nerve supplies the superior, internal and inferior recti, and the inferior oblique muscles of the eyeball, as well as the levator palpebræ superioris, the ciliary muscle, and the constrictor of the iris. The fourth nerve supplies the superior oblique, and the sixth nerve the external rectus muscles. The dilator of the iris is innervated by the sympathetic. Paralysis of the third nerve results in ptosis, wrinkling of the forehead (to compensate for the lost power of the levator palpebræ), divergent strabismus, diplopia, and a fixed and dilated pupil (cycloplegia) inactive to light and accommodation (iridoplegia). The eyeball is pulled outward by the active external rectus, and may be moved slightly downward and inward by the active superior oblique. Paralysis of the fourth nerve results in diplopia, especially when the patient looks downward, and perhaps deficient downward and inward movement of the eyeball (usually slight and unnoticed). Paralysis of the sixth nerve results in deficient movement of the eyeball outward or internal strabismus, and diplopia when the patient looks outward toward the affected side.

The diplopia that results from paralysis of the ocular muscles may be homonymous (simple) when the false image (that seen by the affected eye) is on the same side as the paralyzed eye (usual in convergent strabismus); or the diplopia may be crossed, when the false image is seen on the other side of the true image (usual in divergent strabismus). In addition to restriction of movements of the eyeball, strabismus, and diplopia, paralysis of the ocular muscles usually leads to (*a*) secondary deviation of the sound eye, which may be demonstrated by covering the sound eye and directing the patient to fix on an object with the paralyzed eye toward the direction of paralysis; upon uncovering the sound eye,

it will be seen to have been moved farther in the direction of fixation than the paralyzed eye, on account of the greater response to an equal innervation on the part of the normal muscles of the non-paralyzed eye; and (*b*) erroneous projection of the field of vision.

Occasionally, from overstimulation in hysterical states, in meningitis, etc., spasm of the ocular muscles may be observed. Nystagmus, a clonic, usually bilateral spasm of the ocular muscles, causing involuntary, rhythmic, oscillatory movements of the eyeballs, occurs in Friedreich's ataxia, multiple sclerosis, and other diseases of the nervous system, in nodding spasms of children, in miners, etc.

Ophthalmoplegia.—Disease of the nuclei of the ocular nerves results in the condition called ophthalmoplegia (nuclear ocular palsy), which is discussed on page 851.

V. **Trigeminal Nerve.**—The fifth nerve is made up of motor fibers which supply the muscles of mastication, and sensory fibers which, through the medium of its three great branches (ophthalmic, superior and inferior maxillary), supply half of the face and the anterior portion of the head, the conjunctiva, orbit, eyeball, nose, nasal cavity, lips, teeth, mouth, hard and soft palate, and anterior two-thirds of the tongue. Some observers also believe that the nerve conveys special taste fibers to the anterior two-thirds of the tongue. Paralysis of the motor portion of the nerve gives rise to loss of power of mastication on the affected side, inability to move the jaw toward the sound side (pterygoid muscle), and deviation of the jaw, when depressed, toward the paralyzed side. Irritation of this motor portion gives rise to spasm of the muscles of mastication (trismus), which may be tonic (as in tetanus, tetany, hysteria, reflex local irritation) or clonic (as in "chattering teeth," chorea, etc.). Disease of the sensory portion of the nerve (including the Gasserian ganglion) may give rise to: (1) Neuralgia, which usually involves one of the three great divisions, sometimes all three, and is often intractable (tic douloureux); (2) neuritis and herpes zoster; (3) anesthesia of part or all the area of distribution; and (4) nutritional or trophic disorders, such as lessening of the lacrymal secretions (to which conjunctivitis and ulceration of the cornea may be due) and of the salivary and buccal secretions, facial hemiatrophy, etc. Some observers believe that loss of the sense of taste over the anterior two-thirds of the tongue follows disease of the fifth nerve, but the available evidence is not conclusive.

VII. **Facial Nerve.**—The facial nerve innervates the muscles of the face; perhaps it contains also some sensory fibers (geniculate ganglion and nerve of Wrisberg—Hunt). Paralysis of the nerve may be central or peripheral.

Central Facial Paralysis.—The central case may be due to disease of (1) the nuclei in the pons; this occurs in tumors, hemorrhage, and softening, and is usually associated with disease of adjacent nuclei; or (2) the fibers above the nuclei in the pons, the peduncle, the corona radiata, the internal capsule, or the cortex. This is usually a part of hemiplegia, and is due to like cause—hemorrhage, softening, tumors, abscess, etc.;

rarely the facial nerve may be involved alone (internal capsule or cortical disease). This central paralysis differs from the peripheral type in the common freedom of the upper branches of the nerve (so that the patient can wrinkle the forehead and elevate the eyebrow); in the preservation of the normal electrical reactions of the nerve and muscles; and in the much less impairment of the emotional movements (laughing, crying) as contrasted with the voluntary movements of the facial muscles.

Peripheral Facial Paralysis (Bell's Palsy).—Peripheral facial paralysis may result from (1) disease of the pons, usually tumors, softening, or inflammatory processes; or the paralysis may be part of a hemiplegia, in which event it comprises a so-called crossed paralysis—involvement of the face on the side of the lesion (the facial fibers have already decussated) and involvement of the arm and leg on the opposite side; (2) disease at the cerebellopontine angle, at the base of the brain where the nerve emerges from the pons—usually tumors, gummas, tubercles, meningitis, traumas; (3) disease in the Fallopian canal—caries and inflammation, especially the results of middle-ear suppuration; (4) disease outside the stylomastoid foramen—traumas, tumors of the parotid region, the influence of cold. The cases to which the term Bell's palsy is restricted are commonly attributed to cold and exposure (doubtless some infection). The lesions are inflammatory in nature and are usually most marked within the Fallopian canal, the unyielding wall of which leads to compression and atrophy of the nerve fibers. The symptoms (since the disease is a disorder of the lower motor neuron) consist of paralysis of muscles innervated by the nerve, together with wasting and fibrillary twitchings of the muscles and the electrical reactions of degeneration. The face becomes flattened, the nasal fold obliterated, the angle of the mouth drooped (that of the other side looks and may become retracted), and there is inability to wrinkle the forehead, to frown, to close the eyelids, to show the teeth, etc. There may also be impairment of hearing (branch nerve to the stapedius), and of taste over the anterior two-thirds of the tongue (chorda tympani). The cutaneous sensibility is preserved. Complete recovery is unusual; secondary contractures are common. The treatment comprises the use of hot applications, counter-irritation, massage, faradization, the iodides, etc.; the treatment proper to any local disorder that may be present; and in cases not soon improved (three to four months) anastomosis of the spinal accessory or the hypoglossal with the facial nerve.

Facial Spasm.—Facial spasm may be an organic disorder or it may arise reflexly from irritation due to carious teeth, for instance. The spasm may involve some or all the muscles supplied by the facial nerve, and may be unilateral or bilateral. Usually the muscles about the eye, or these and those of the side of the face, reveal rapidly recurring twitchings. The disorder is painless, although the nerve may have various tender spots. Occasionally, as after paralysis, tonic spasms of the facial muscles may ensue. Most of the cases of facial spasm last many years,

and are scarcely curable, although benefit may follow removal of sources of irritation, counterirritation, surgical intervention, etc.

VIII.—**Auditory Nerve.**—The eighth nerve consists of two parts, the cochlear nerve, which connects the organ of Corti in the cochlea with the temporosphenoidal lobe and is the nerve of hearing; and the vestibular nerve, which connects the vestibule and the semicircular canals with cerebellar and other centres and serves to maintain equilibrium.

Cochlear Nerve.—Disorder of the cochlear nerve gives rise to tinnitus aurium; sometimes to hyperacute or painful hearing (hyperacusis); most often to nervous deafness. If a tuning-fork is not well heard when held near the auditory meatus, but is well heard when placed on the temporal bone (the vibrations being transmitted through the bone to the cochlea and vestibule), the deafness is probably not nervous (not due to disorder of the nerve).

Vestibular Nerve; Meniere's Disease.—Disorder of the vestibular nerve is the cause of so-called *auditory vertigo*, or *Ménière's disease*, a symptom complex, consisting of tinnitus aurium, vertigo, nausea, and vomiting, developing suddenly and sometimes attended by nervous deafness. The paroxysms occur irregularly—at long or short intervals. In addition, there may be nystagmus, diplopia, ataxia, etc. Rarely the disease is of the apoplectiform type, due to hemorrhage in the labyrinth; usually it occurs in those the subjects of acute or chronic ear disease—of the Eustachian tube, the middle ear, or the internal ear; it may be due to angioneurotic disturbances in the labyrinth; or attacks may be provoked by cerumen in the auditory canal, syringing the ear, etc. The prognosis in Ménière's disease is not good. Relief may be afforded by treating any local source of irritation, correcting refractive errors, the use of bromides, nitroglycerin in the event of arteriosclerosis and high blood pressure, etc.

IX.—**Glossopharyngeal Nerve.**—The ninth nerve supplies motor fibers to the stylopharyngeus muscle and perhaps the palate, sensory fibers to the upper part of the pharynx, tonsil, soft palate, etc., and taste fibers to the posterior third of the tongue. The nerve scarcely, if ever, is diseased alone, being intimately related to the vagus. However, impairment of the sense of taste on the posterior third of the tongue and difficulty in swallowing may be attributed to it.

X. **Pneumogastric or Vagus Nerve.**—The tenth nerve supplies the pharynx, larynx, lungs, heart, œsophagus, and stomach, and is intimately associated with the glossopharyngeal, spinal accessory, hypoglossal, and sympathetic nerves. Disease of the nuclei of these nerves constitutes bulbar paralysis. In paralysis of the pharyngeal branches of the vagus (together with the glossopharyngeal) there is difficulty in swallowing, food is not passed on to the œsophagus, and may regurgitate through the nostrils. Spasm of the pharyngeal muscles may be seen in hysterical subjects, and is the obtrusive feature of hydrophobia. Paralyses of the laryngeal branches of the vagus causes bilateral paralysis (nuclear disease) or unilateral paralysis (usually nerve disease) of the laryngeal

muscles. Bilateral paralysis of the abductors (posterior crico-arytenoids) occurs in central disease (tabes, bulbar paralysis), pressure on the nerves, hysteria, etc., and is characterized by inspiratory stridor, more or less disordered phonation, and the cadaveric position of one or both vocal cords. Unilateral abductor paralysis follows involvement of one recurrent laryngeal nerve—usually pressure due to an aneurysm of the aorta, and is characterized by hoarseness of the voice and a brassy cough. Adductor paralysis is occasionally seen in hysteria; the vocal cords are not approximated during attempts at phonation, and the voice is lost. Spasm of the adductor muscles may occur in the event of central or peripheral irritation, and is a marked feature of laryngismus stridulus in rickety children. The cardiac branches of the vagus regulate and inhibit the action of the heart; irritation results in slowing of the heart beat, and paresis or paralysis (removal of inhibiting influence) results in acceleration of the beat. The subjective sensations of the heart beat are conveyed to the brain by the vagus. Little is known regarding disorder of the pulmonary branches of the vagus, although these are said to influence the bronchial musculature and are perhaps in some way concerned in the paroxysms of bronchial asthma. Spasm of the œsophagus, of the cardia, and of the pylorus, vomiting, the gastric crises in tabes, and other gastro-intestinal disturbances are also related to disorder of the vagus.

XI. **Spinal Accessory Nerve.**—The eleventh nerve, in part, joins the vagus nerve and is distributed with it; other fibers innervate the sternomastoid and the trapezius muscles. Paralysis of this part of the nerve results in paralysis of the sternomastoid muscle and inability to rotate the head to the opposite side, and of the trapezius muscle and lessened ability to elevate the shoulder, and the arm above the horizontal position. Complete paralysis of the trapezius does not ensue, since it is innervated in part by the cervical nerves. Spasm of the accessory nerve results in wryneck (torticollis). This may be a congenital (permanent) affection, due to shortening of the sternomastoid muscle (usually the right), and is often associated with facial asymmetry. An acquired, spasmodic wryneck is common in women, especially hysterical subjects; it has been attributed to cold. The spasms may be tonic or clonic. When the sternomastoid is affected alone, the head is rotated to the opposite side, the chin somewhat tilted upward, and the occiput somewhat depressed; when the trapezius is also involved, the head is still further depressed toward the shoulder, and not so far rotated. The contracted muscles stand out as prominent tense cords. There is usually complaint of pain and stiffness of the neck. The cases that develop suddenly after a cold usually recover promptly under the influence of hot applications, massage, and the salicylates internally. In the other cases the outlook is not so good and relapses are common. Relief may attend the use of the bromides, galvanism, mechanical fixation of the head, section of the contracted muscles, division of the accessory nerve, etc.

XII. **Hypoglossal Nerve.**—The twelfth is the motor nerve of the tongue. The tongue is usually involved in hemiplegia (cortical or supra-

nuclear lesion). Nuclear lesions occur in bulbar paralysis, tabes dorsalis, syringomyelia, tumors, etc., and unilateral disorder may follow tumors, meningitis, etc. The chief symptoms are unilateral or bilateral paralysis, atrophy, and fibrillary twitchings of the tongue; the mucous membrane of the tongue becomes wrinkled; and difficulty is usually experienced in pronouncing certain words. Spasm of the tongue is unusual, but may be encountered in hysteria, epilepsy, chorea, stuttering, etc.

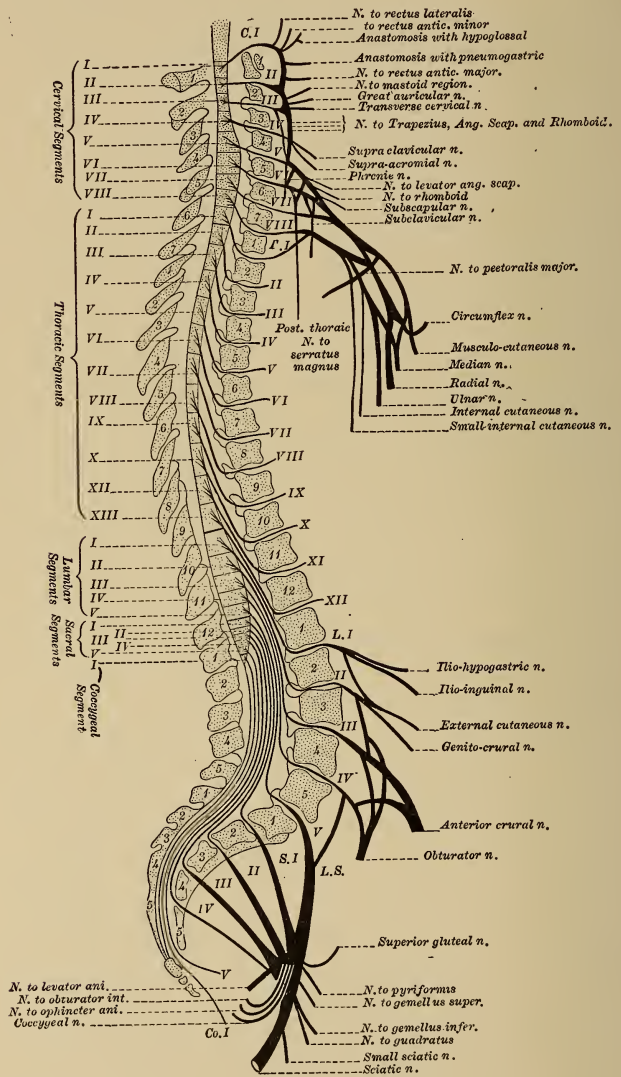
DISEASES OF THE SPINAL NERVES.

The Cervical Plexus.—Cervicoöccipital neuralgia, involving the area of distribution of the first four cervical nerves (the occipitoparietal region), may be due to the causes of neuralgia mentioned elsewhere (page 808), or to local disease of the bone, the pressure of tumors, etc. The phrenic nerve, derived from the third, fourth, and fifth cervical roots (Fig. 16), may be paralyzed, in consequence of disease of the spinal cord (anterior horns), meninges, or vertebræ; of compression, as by a tumor; or of neuritis, etc. The symptoms consist of paralysis (immobility) of the diaphragm (unilateral or bilateral, as the case may be); immobility or retraction of the upper abdomen during inspiration, and, perhaps, bulging during expiration; and dyspnœa upon exertion. The inactivity of the diaphragm may be readily recognized by radiographic examination. The inaction due to inflammatory conditions, and the depression due to pleural effusion, emphysema, etc., must be excluded. Spasm of the diaphragm may be due to inflammatory or other irritative (sometimes so-called neurotic) conditions of the diaphragm or phrenic nerve; and when intermittent, is shown by hiccough.

The Brachial Plexus.—The brachial plexus is made up of the fifth, sixth, seventh, and eighth cervical and the first thoracic roots. Paralysis may be due to disease of the spinal meninges or the vertebræ; tumors and other compressing disorders of the neck; traumatism, such as fracture or dislocation of the vertebræ; stretching and laceration of the nerves; or neuritis. Since the root fibers combine variously to form the peripheral nerves, the clinical manifestations of a root lesion differ from those of a peripheral nerve lesion. The important clinical conditions comprise: (1) *Obstetrical or birth paralysis* (*upper arm type of Erb*), which results from laceration usually of the fifth and sixth, sometimes of all the roots, due to forcible stretching apart of the head and shoulder during birth. Usually the deltoid, biceps, supraspinatus, infraspinatus, brachialis anticus, and supinator longus are paralyzed and atrophied, but in some cases complete motor and sensory paralysis of the arm may occur (laceration of the entire plexus). (2) *Lower arm type*, due to involvement of the seventh and eighth cervical and the first thoracic roots. This is followed by paralysis of the triceps, pronators and flexors of the wrist, the flexors and extensors of the fingers, and the small muscles of the hand. Paralysis of the small muscles of the hand, anesthesia in

the ulnar distribution, and ocular symptoms, due to disorder of the cervical sympathetic, together comprise *Klumpke's paralysis.* (3) *Cer-*

FIG. 16



The relations of the segments of the spinal cord and their nerve roots to the bodies and spines of the vertebræ. (Starr.)

vical rib. An accessory cervical rib, usually compressing the seventh or eighth cervical roots or the subclavian artery, is a not uncommon

cause of pain and paresthesia of the arm and hand, spasm and atrophy of the small muscles of the hand, local asphyxia, and even gangrene of the fingers. The condition may be familial, and may not become apparent until adult life. It is readily recognized upon radiographic examination, and is curable upon removing the accessory rib.

Lesions of the individual nerves of the brachial plexus give rise to the following phenomena: (1) The circumflex nerve is often injured in dislocation of the shoulder and other injuries, etc., and leading to paralysis and atrophy of the deltoid prevents raising the arm to the horizontal position. (2) The long or posterior thoracic nerve, which fixes and (with the trapezius) rotates the scapula, when paralyzed gives rise to a "winged" scapula, or prominence of the vertebral border (serratus paralysis), and imperfect elevation of the arm above the shoulder (lessened rotation of the scapula). (3) The musculospiral nerve is often disordered—in consequence of cold, injuries, fracture of the humerus, etc.; of pressure, as by falling asleep with the arm on the back of a chair, counter, or bar (bartender's paralysis); or as may occur in so-called "bridal" paralysis and other paralyses resulting from a weight; and of lead poisoning and other forms of intoxication. Usually the lesion is just above the elbow, and results in paralysis of the extensor muscles of the wrist and the fingers (except the supinator longus), and the characteristic wrist-drop. In lesions higher up, the triceps, the brachialis anticus, and the supinator longus are also involved. (4) The ulnar nerve, when paralyzed, results in atrophy and loss of power of the interossei, all the muscles of the little finger, the ulnar half of the deep flexors of the fingers, the adductor and the inner head of the short flexor of the thumb, and the ulnar flexor of the wrist. The conspicuous symptoms consist of impaired flexion of the hand, loss of power of abducting and adducting the fingers, and of flexing and extending the second and third phalanges. There is also anesthesia of both surfaces of the fourth and of the ulnar half of the third fingers. In long-standing cases the claw-like hand (main-en-griffe) develops, being due to the activity of the non-opposed antagonistic muscles supplied by the median nerve. (5) The median nerve is rarely involved alone, but if so, it may lead to paralysis of the muscles arising from the inner condyle of the humerus (except the flexor carpi ulnaris) and the deep muscles of the forearm; in consequence, the arm cannot be pronated beyond the mid-position, the wrist cannot be flexed toward the radial side, the second phalanges cannot be flexed, and the thumb cannot be opposed to the finger tips. There is also anesthesia of both surfaces of the thumb, and the first, second, and the radial half of the third finger. (6) *Volkmann's contracture* is a form of paralysis of the hand and forearm that usually follows fractures about the elbow or of the forearm and undue pressure caused by splints and tight bandaging. The nature of the disease is not definitely known; it has been attributed to defective blood supply (whence ischemic paralysis), to primary disorder of the muscles (myositis and fibroid changes), to neuritis, and to changes

following sloughing of the tissues of the forearm (which is sometimes observed). The symptoms consist of atrophy of the muscles of the forearm and hand, with contraction of the flexors, and the ultimate development of the claw-like hand. Usually there are no noteworthy sensory changes. The condition may be relieved by passive movements, massage, electricity, or operative measures.

The Lumbar Plexus.—The lumbar plexus is made up of the first three lumbar roots and a branch from the fourth. The chief causes of disorder of the plexus are regional tumors, disease of the vertebræ, psoas abscess, and neuritis; and the obturator nerve may also be injured during parturition or by other trauma. The main symptoms consist of deficient adduction and rotation of the thigh (inability to cross the leg over the other). Paralysis of the anterior crural nerve results in paralysis and atrophy of the extensors of the knee, loss of the knee-jerk, and anesthesia of the anterolateral aspect of the thigh and of the inner aspect of the leg. Disease of the external cutaneous nerve may result in *meralgia paræsthetica*, a paresthesia of the outer aspect of the thigh.

The Sacral Plexus.—The sacral plexus is made up of a branch from the fourth lumbar, the fifth lumbar, the first, second, and third sacral, and a branch from the fourth sacral roots. The chief causes of disorder of the plexus are pelvic growths and inflammations, disease of the vertebræ and the spinal meninges, parturition, and neuritis. Involvement of the sciatic nerve causes sciatica (page 809). Paralysis of the nerve results in inability to flex the leg upon the thigh and loss of power of all the muscles below the knee; anesthesia of the outer aspect of the leg and of the sole and most of the dorsum of the foot; and ultimate wasting of the muscles. Paralysis of the external popliteal (or peroneal) nerve results in loss of power of the peroneal muscles, the extensors of the toes, and the tibialis anticus, with ensuing "foot-drop" and a characteristic, so-called, steppage gait. Paralysis of the internal popliteal nerve results in loss of plantar flexion of the foot and of the power to flex the toes, and anesthesia of the outer and posterior aspect of the lower leg and sole of the foot.

DISEASES OF THE CERVICAL SYMPATHETIC NERVES.

The cervical sympathetic system supplies vasomotor, secretory, and motor-nerve fibers to the head and neck and part of the arm and chest: vasomotor fibers to the bloodvessels, secretory fibers to the sweat glands and the submaxillary salivary glands, and motor fibers to the dilator muscle of the iris, the involuntary muscle fibers of the upper eyelids, and the orbital muscle of Müller. The sympathetic nerves may be paralyzed or irritated, paralysis being much the more common. The symptoms of paralysis consist of unilateral ptosis, or narrowing of the palpebral fissure (paralysis of the unstriped muscle of the upper eyelid); enophthalmos or retraction of the eyeball (paralysis of Müller's muscle);

contraction of the pupil and no dilatation when it is shaded (unopposed action of the sphincter muscle of the iris supplied by the intact third cranial nerve, which is also responsible for preservation of contraction of the pupil to light and in accommodation); unilateral pallor and lessened secretion of sweat (anidrosis); and perhaps dryness of one nostril and one-half of the mouth. Irritation of the sympathetic nerves causes contrary symptoms: widening of the palpebral fissure, protrusion of the eyeball (exophthalmos), dilatation of the pupil, flushing of the skin, and increased sweating (hyperidrosis).

DISEASES OF THE CEREBROSPINAL MENINGES.

MENINGEAL HEMORRHAGE.

Etiology.—Meningeal hemorrhage may be extradural or intradura (subdural). Extradural cerebral hemorrhage is almost always traumatic, though it may result from erosion of a vessel by caries of the bone. The middle meningeal artery is usually, but not always, the source of the hemorrhage. Intradural cerebral hemorrhage (hemorrhage from the vessels of the pia-arachnoid) may be due to traumatism (rather unusual), internal hemorrhagic pachymeningitis and associated disorders of mentality, rupture of an aneurysmal dilatation of a vessel, the hemorrhagic diathesis, severe anemic states, paroxysms of whooping cough, the chronic congestion of cardiac, pulmonary, and advanced renal diseases; and it may occur from compression of the skull at birth. Spinal meningeal hemorrhage may or may not be associated with cerebral hemorrhage; when it occurs it may be due to like causes; or it may result from local traumatism, and is then usually extradural.

Symptoms.—The symptoms vary, depending upon the situation of the hemorrhage, upon whether it is small or excessive, and whether it occurs slowly or rapidly. In sudden, large, *cerebral meningeal hemorrhage* the symptoms are much like those of intracerebral apoplexy, with the added evidences of cortical irritation or paralysis. In other cases the manifestations of the injury itself sometimes overshadow those of the hemorrhage, especially if the hemorrhage occurs slowly and is not directly over the motor area. In this event the patient may even continue at his occupation for several hours, perhaps complaining only of headache; in other cases, there may be also some mental confusion, vertigo, and vomiting, and the patient gradually loses consciousness. Sometimes convulsions, which may be severe, occur; they may be unilateral and succeeded by paresis or paralysis (monoplegia or hemiplegia). In other cases, depending upon the seat of the hemorrhage, other symptoms develop, such as aphasia, sensory disturbances, oculomotor palsy (occipital region), etc. In favorable cases the coma lessens and partial recovery of the palsy supervenes.

The symptoms of *spinal meningeal hemorrhage* consist of pain and distress in the back, which are usually of sudden onset (in traumatic cases especially); radiation of the pain along the nerve roots and peripheral nerves, especially into the legs; and some muscular spasm. The deep reflexes may be temporarily lessened; usually they become exaggerated. In the event of compression of the cord, more or less paralysis supervenes and control of the vesical and rectal sphincters may be lost.

Treatment.—The treatment of cerebral meningeal hemorrhage is similar to that of intracerebral hemorrhage; but in every case the head should be shaved and searched for subcutaneous hemorrhage or fracture, and the question of operative removal of the clot must be considered. When in doubt, it is probably wise to operate, since a large hemorrhage may result in death from compression, and a small hemorrhage, if recovered from, may determine traumatic epilepsy or insanity in later life. In spinal meningeal hemorrhage resort should be had to lumbar puncture, with a view to relieve pressure; but if symptoms of compression of the cord ensue, laminectomy should be performed and the fluid and clotted blood removed; otherwise, the treatment consists of rest and quiet.

EXTERNAL PACHYMENINGITIS.

Etiology.—Inflammation of the outer surface of the dura mater is practically always a secondary affection, and is usually acute, although it may be chronic. It results from trauma (injuries to the skull or vertebræ) or bacterial infection—most commonly in consequence of necrosis of the petrous portion of the temporal bone in middle-ear disease, or of streptococcic or staphylococcic, less commonly of tuberculous or syphilitic, disease of the bones or adjacent tissues. The chronic cases are virtually always due to syphilis or tuberculosis.

Pathology.—In acute cases the dura is swollen, softened, hemorrhagic, and frequently suppurative. The pus collects between the membranes and the skull or vertebræ, and may extend into and between the two layers of the membrane (causing thrombosis of the sinuses), and involving the pia-arachnoid. In syphilitic and tuberculous cases the dura may be greatly thickened and considerable pus may be present, although the inner surface may be quite smooth. In chronic pachymeningitis the lesions consist of the formation of the new fibrous tissue.

Symptoms.—The symptoms in mild cases are indefinite; in the cerebral cases there may be only headache. In severe cases the symptoms of the primary disturbance often mask the pachymeningitis. Symptoms, when present, consist of headache, malaise, and perhaps convulsions, followed by drowsiness and stupor, and in case of suppuration, by chills and fever, and often sweats and leukocytosis. Sometimes the symptoms are only those of pressure, which may be sufficient to cause paralysis of the opposite side of the body. Choked disk has also been noted. Sinuses communicating with the exterior usually suggest

syphilis. The symptoms in the spinal cases are those of compression of the spinal cord, which are usually due, in part, also to associated lesions.

Treatment.—The treatment varies with the cause. In the lighter grades of the disease, antiphlogistic measures and counterirritants are indicated; in the severer grades, operative interference; and in syphilitic cases, mercury and potassium iodide.

INTERNAL PACHYMENINGITIS.

Three forms of internal pachymeningitis are usually described: The hemorrhagic, the purulent, and the pseudomembranous. The purulent and the pseudomembranous forms are uncommon and rarely recognized before death. The purulent form is usually secondary to inflammation of the pia-arachnoid, but it may follow softening of a sinus thrombus or result from traumatism. The pseudomembranous form is of obscure etiology, though it may result from trauma, or syphilis, or follow hemorrhagic or suppurative pachymeningitis. The lesions may be purely fibrous or partly osseous.

Internal hemorrhagic pachymeningitis may involve the cerebral or the spinal meninges. The lesions are most common in the brain (hematoma of the dura mater), and occur chiefly in males over fifty years of age, though they are by no means rare in children. The most frequent causes are: (1) Chronic alcoholism; (2) different nervous disturbances, such as paretic dementia, chronic hereditary chorea, senile dementia, etc.—whence it is frequently seen in insane asylums and almshouses; (3) cranial injuries; (4) different infectious diseases; (5) diseases of respiration, of circulation, and of nutrition.

Pathology.—The lesions are usually confined to the area of distribution of the middle meningeal artery and are often bilateral; they consist of the development of a very delicate vascular membrane (the result of the organization of an inflammatory exudate), the bloodvessels of which commonly rupture and give rise to the extravasation of a greater or less amount of blood (hematoma). Through the successive formation of such vascular membrane and the occurrence of hemorrhage, lamellæ of membrane and hemorrhage are formed, which may be 3 to 5 mm. thick. The lamellated structure is well shown on section, and frequently the successive layers may be stripped off. The brain is correspondingly compressed. Occasionally, in consequence of the imperfect organization of the blood clot, a collection of serous fluid or a cyst results (hygroma dura matris). Involving the spinal meninges, the lesions are most common in the cervical region—hypertrophic cervical pachymeningitis. A new formation, analogous to that in the brain, develops between the dura and the spinal cord, compressing the cord and the nerve roots, and often leading to secondary degenerations.

Symptoms.—In many cases of the *cerebral* disorder symptoms are wanting, especially when the hemorrhage is small and has occurred slowly; and in the insane, in whom the insanity often masks the symp-

toms of the pachymeningitis. Characteristic symptoms, however, consist of recurring apoplectiform attacks (referable to repeated hemorrhages), evidences of increasing intracranial pressure, and paresis or paralysis of cortical origin. Headache is common. The symptoms of increasing intracranial pressure consist of increasing headache, somnolence, progressing in some cases to coma, nausea, vomiting, slowing and irregularity of the pulse, and contracted pupils (with marked or complete loss of reaction to light). Paralysis of one limb, or of a single cranial nerve (such as the facial or hypoglossal), and circumscribed or unilateral convulsions, suggest the cortical seat of the disorder. The patient often recovers from minor attacks, the manifestations of the apoplectiform insult gradually (within several weeks) subsiding, though paresis or paralysis, headache, intellectual disturbances, muscular weakness and awkwardness, etc., often persist. In severe cases the manifestations are often bilateral, and the patient may die in coma—an event that happens sooner or later in all cases, whether the lesions are unilateral or bilateral.

The symptoms of the *spinal* disorder consist of severe neuralgic pains in the neck and arms (corresponding to the cervical roots involved), with associated hyperesthesia, paresthesia, and perhaps anesthesia. In the course of time muscular weakness and atrophy supervene; these depend upon the situation and extent of the nerve-root involvement, but are usually most marked in the small muscles and the flexors of the hands. The extensors often remaining intact, gradually lead to the development of the claw-like hand. In some cases the hands, forearms, upper arms, and shoulders may be markedly involved (cervical paraplegia). Ultimately spastic paraplegia may develop in the legs from secondary degeneration in the cord.

Diagnosis.—The diagnosis of the cerebral disorder is always difficult; it is suggested by the etiological factors and recurring apoplectiform attacks, with evidences of cortical irritation. The spinal disorder must be differentiated from tumors and other forms of compression, amyotrophic lateral sclerosis (absence of severe initial pains and sensory changes), and syringomyelia (characteristic sensory changes).

Prognosis.—The outlook in both types of the disorder is bad.

Treatment.—The causal condition demands first attention, and the symptoms should be treated as they arise: during the stage of hemorrhage—rest in bed, elevation of the head, an ice-cap or leeches to the base of the head or behind the ears, hot foot baths, venesection, free purgation, etc.; and during the subsequent stages—mercurials, potassium iodide, and antispasmodics. Should the diagnosis of a focus of cortical irritation be possible, especially with evidences of increasing intracranial pressure, an operation for the removal of the clot may be undertaken.

LEPTOMENINGITIS.

Etiology.—Inflammation of the pia-arachnoid (leptomeningitis, pia-arachnitis) may be primary or secondary, and acute or chronic. The

primary acute form, even the sporadic cases, is commonly the cerebrospinal fever (meningococcic infection) already discussed (page 86), although tuberculous and pneumococcic infections also may occasion lesions only, or most marked, in the meninges. Secondary leptomeningitis may be due to: (1) Bacterial infection, and is seen most commonly, as a complication or sequel, in the acute infectious diseases, especially in pneumonia, septicemia, erysipelas, acute infective endocarditis, typhoid fever, smallpox, measles, scarlet fever, gonorrhœa, influenza, acute rheumatic fever, lung abscess, empyema, etc. It results also from extension of infection from caries of adjacent bones, from purulent otitis media, disease of the frontal sinuses, nasal cavities, or pharynx, brain abscess, etc. (2) Trauma of the bones of the skull, affecting the meninges directly; but in most of these cases, trauma and infection are associated. (3) As a terminal infection in many chronic diseases, such as nephritis, diabetes, arteriosclerosis, etc. The exciting causes may be the microörganisms provocative of the primary infection, but often they are the common pyogenic bacteria, the infection of the meninges being hemogenic, lymphogenic, or occurring by contiguity of tissue.

Pathology.—The lesions vary much in different cases. Basal meningitis, convexity meningitis, and ventricular meningitis are terms used to denote the seat of the marked lesions; but in many cases the lesions are more or less widespread. In the meningitis following pneumonia and sepsis the convexity on both sides is usually involved; in that occurring in children the posterior basic region is especially involved (posterior basic meningitis), and this is also true of that occurring in Bright's disease, diabetes, and other cachectic conditions. Anatomically, three varieties may be distinguished—the serous meningitis of Quincke, tuberculous meningitis (page 122), and purulent meningitis.

The *serous meningitis of Quincke* is a rare condition, in which the pia-arachnoid is the seat of serous inflammation, especially marked on the convexity, and in which, by lumbar puncture, the cerebrospinal fluid is found under greater pressure than the normal 40 to 60 mm. of water, and it contains at least twice as much as the normal (0.5 per cent.) of albumin.

Generally, however, the lesions are purulent and confined to, or at least most marked on, the convexity of the brain (convexity meningitis). In some cases, especially in infants or children, they are more marked at the base (posterior basic meningitis). In other cases the lesions are widespread, and consist of swelling, congestion, and suppuration of the pia-arachnoid, sometimes its more superficial layers, especially marked along the course of the vessels, and sometimes so marked as completely to obscure the convolutions. They are usually bilateral, although they may be unilateral, especially in cases due to extension from middle ear disease, etc., when they may be accompanied by sinus thrombosis. The ventricles may contain, and are sometimes distended with, serous or seropurulent fluid; the lesions may extend to, or simultaneously involve, the spinal meninges (cerebrospinal meningitis); and in some cases small foci of

hemorrhage may be detected in the superficial layers of the brain (meningo-encephalitis).

Chronic leptomeningitis is quite uncommon; it may be due to syphilis or tuberculosis; rarely the meningococcic (epidemic or sporadic) cases, posterior basic meningitis, and the serous meningitis of Quincke may run a chronic course.

Symptoms.—The symptoms are sometimes divided into the prodromal, the irritative, and the paralytic. Prodromal symptoms are frequently absent, but sometimes for a day or two there may be general malaise, irritability, headache, and perhaps vomiting; but these are often overshadowed or masked by the evidences of the primary disorder. The disease is often ushered in with high fever, perhaps a chill or chilliness, and headache which is extreme, persistent, sometimes more marked in the frontal region, but usually widespread—constituting a most characteristic symptom of the disease. With the progress of the inflammation, the headache increases in severity, and is often subject to exacerbations of great severity (occasioning the so-called "hydrocephalic cry"), and disorders of consciousness—delirium, stupor, coma—supervene. Furthermore, manifestations of cerebral irritation develop, and vary somewhat with the seat of the most marked lesions; they comprise vomiting (often frequent and persistent, especially in basilar meningitis); slowing and irregularity of the pulse; retraction of the head and rigidity of the neck muscles (opisthotonos), especially when the lesions are posterior, and basal, and cervical; crunching of the teeth; pupillary miosis; strabismus; nystagmus; photophobia; undue sensitiveness of the auditory apparatus; ptosis; muscular irritability and twitchings; convulsive seizures in the facial muscles (trismus) and in the extremities; increase of the reflexes; Kernig's sign (inability to extend the leg when the thigh is flexed on the abdomen); general cutaneous and muscular hyperesthesia; etc. These are soon followed by evidences of paresis or paralysis (paralytic stage): coma instead of the previous delirium and stupor; increased frequency of the pulse (not constant); pupillary differences or mydriasis; sometimes optic neuritis (basilar meningitis); paralysis of certain cranial nerves (especially the facial) or paresis of the extremities of cortical nature; loss of reflexes; retraction of the abdomen; loss of control over the sphincters; etc. Fever is present throughout the course of the disease; it is usually irregular, varying up to 102° or 103° F., but occasionally hyperpyrexia is observed, and sometimes but little fever (in terminal infections). Repeated chills are not uncommon. Herpes labialis is quite common, but especially in the meningococcic infections. The urine is diminished and often contains albumin and casts; the bowels are constipated, until the final stage, when incontinence often supervenes. Leukocytosis is usually present.

Diagnosis.—The diagnosis is usually comparatively easy, but in doubtful cases the antecedent history, disordered cerebration, high fever, slow pulse, and the other symptoms narrated, together with the data mentioned on page 89, should clear up the diagnostic uncertainty.

Prognosis.—The outlook depends upon the nature of the infection, the severity of the lesions, and the associated conditions. Quincke's serous meningitis may eventuate in recovery; the purulent cases are almost always fatal; and the non-fatal cases may be followed by divers sequels (page 89).

Treatment.—The treatment is that mentioned on page 89. Perhaps in the future we may secure a serum or bacterin for each specific type of meningitis.

DISEASES OF THE BRAIN AND SPINAL CORD.

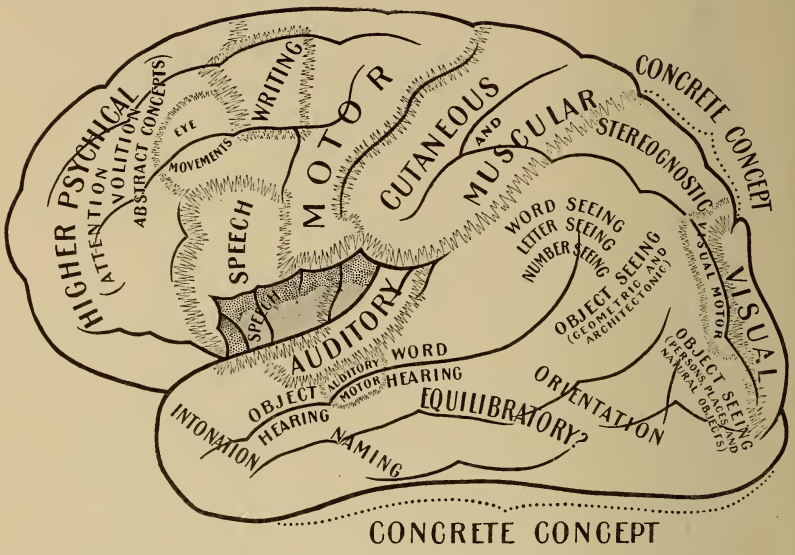
CEREBROSPINAL LOCALIZATION.

Diseases of the brain and the spinal cord may be somewhat arbitrarily divided into two classes: (1) The so-called system diseases—those in which the lesions are more or less strictly limited to certain tracts or collections of neurons functionally homologous; if only one tract is involved, the disorder is spoken of as a single system disease; if more than one, as a combined system disease. (2) The non-system (focal or diffuse) diseases—those in which the lesions are not limited to a system or systems of neurons. The system diseases involve variously the different motor and sensory neurons, of which mention is made on page 802 and thereafter, and which are shown in their course in the spinal cord on Plate II. (page 805).

The accurate localization of irritative or destructive, system or non-system, disorders of the brain and spinal cord depends upon a knowledge of the functional activity and the mutual relations of the different parts of the brain and the cord. The characteristic features of disease of the upper and the lower motor neuron and of the sensory neuron are mentioned on pages 802, 803, and 804. When diseased focally, that is, when the seat of non-system diseases, most parts of the brain and cord exhibit sensible evidence of the local lesions that permit of definite localization; but there are certain so-called silent areas in the brain, the functions of which are unknown, and disease (especially tumors) of which, although causing general manifestations, provoke no localizing symptoms. In the diagnosis of local lesions, especially tumors of the brain, one must remember: (1) That focal signs are of the greater value the nearer to the general symptoms in point of time they develop; (2) that some of the signs are often due to irritation or paralysis of adjacent areas of the brain or of tracts that may pass through or near the seat of disease, and that this is more likely to be the case the longer after the onset of the general symptoms these localizing signs develop; and (3) that in advanced cases many of the signs are often due, not so much to the primary lesion (tumor, abscess, etc.) as to secondary conditions, such as meningitis, hydrocephalus, secondary tumors or abscesses, hemorrhage into tumors, and so forth.

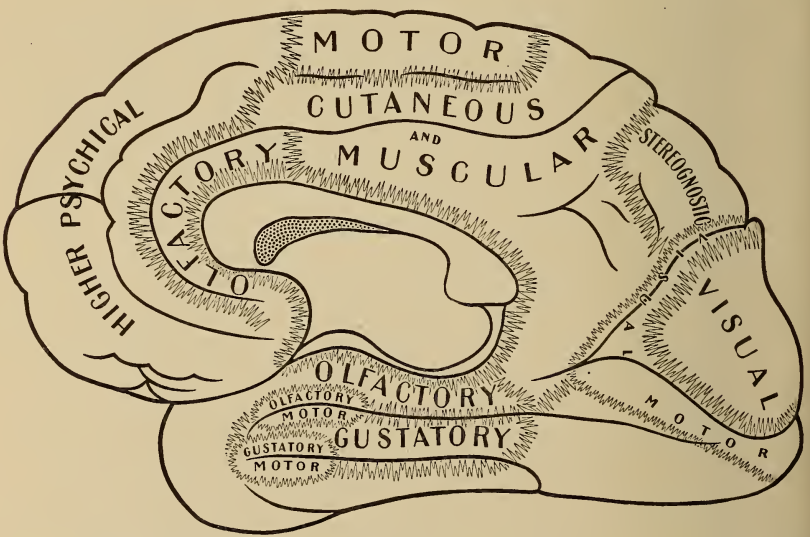
The Brain.—Frontal Lobes; Prefrontal Region.—The frontal lobes anterior to and above the ascending and the third frontal convo-

Fig. 17



The lateral surface of the human brain, showing the localization of the different functions. (Mills.)

Fig. 18



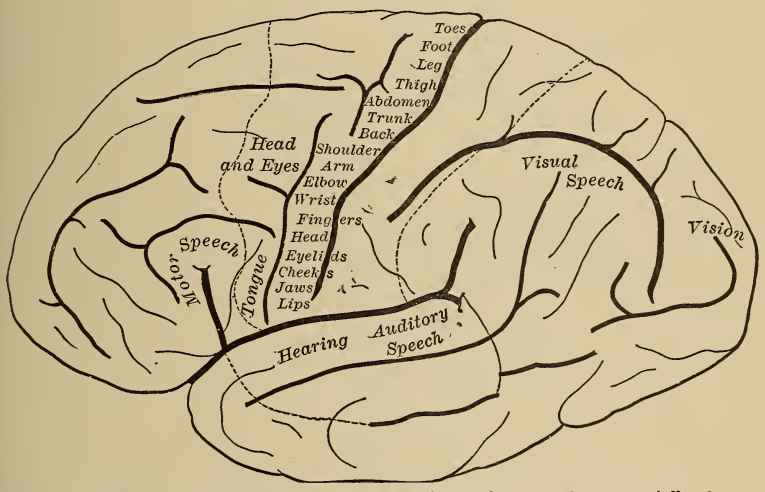
The mesial surface of the human brain, showing the localization of the different functions. (Mills).

lutions are believed to subserve the higher mental and psychic functions, such as general intelligence, memory, reasoning powers, judgment,

attention, volition, and abstract concepts (Figs. 17 and 18). Mills and other observers assign these functions especially to the left side in right-handed persons, and to the right side in left-handed persons. Tumors and other destructive lesions of the prefrontal region are said by Mills to be always associated with disorder of these higher mental functions.

Motor Cortex; Ascending Frontal Convolutions.—The cortical centres for voluntary muscular movements are situated in the ascending frontal, second frontal, and paracentral lobules, the different centres being as represented in Fig. 19. A tumor or other lesion of the motor cortex, or immediately subjacent thereto (subcortical), may produce, first, irritation of the area involved and spasm or convulsions of the muscles of the opposite side of the body therein represented (focal or Jacksonian epilepsy). Soon localized paresis or paralysis supervenes,

Fig. 19



The functional areas of the cerebral cortex of the left hemisphere, to show especially the cortical centres for voluntary muscular movements. (Starr.)

and if at first limited to certain groups of muscles, may, as the tumor grows, become monoplegic or hemiplegic; it usually becomes spastic in type (degeneration of the upper motor neuron). In addition, there is likely to be disorder of epicritic sensibility, especially lessening of tactile sensibility and of the power of cutaneous localization. This is usually due to pressure or secondary involvement of the ascending parietal convolution where sensibility has its cortical representation. Homologous muscles that act together are not likely to show marked or permanent disability from unilateral cortical or subcortical lesions, since the muscles of both sides may be innervated from the one cerebral hemisphere.

Sensory Cortex; Parietal Lobe.—The cortical centres for sensation are situated in the parietal lobe, especially in the ascending

parietal convolution and the gyrus fornicatus. According to Mills, these centres correspond with, and are about on a level with, the related motor centres in the neighboring frontal convolutions. Tumors and other destructive lesions of the parietal lobe causes disturbances of epicritic, protopathic, and deep (or muscular) sensibility—that is, disorder of the sensations of touch, pain, temperature, and stereognosis. Usually also there are motor disturbances from compression by or extension of the tumor to the frontal convolutions, or subcortical implication of the motor fibers in the corona radiata.

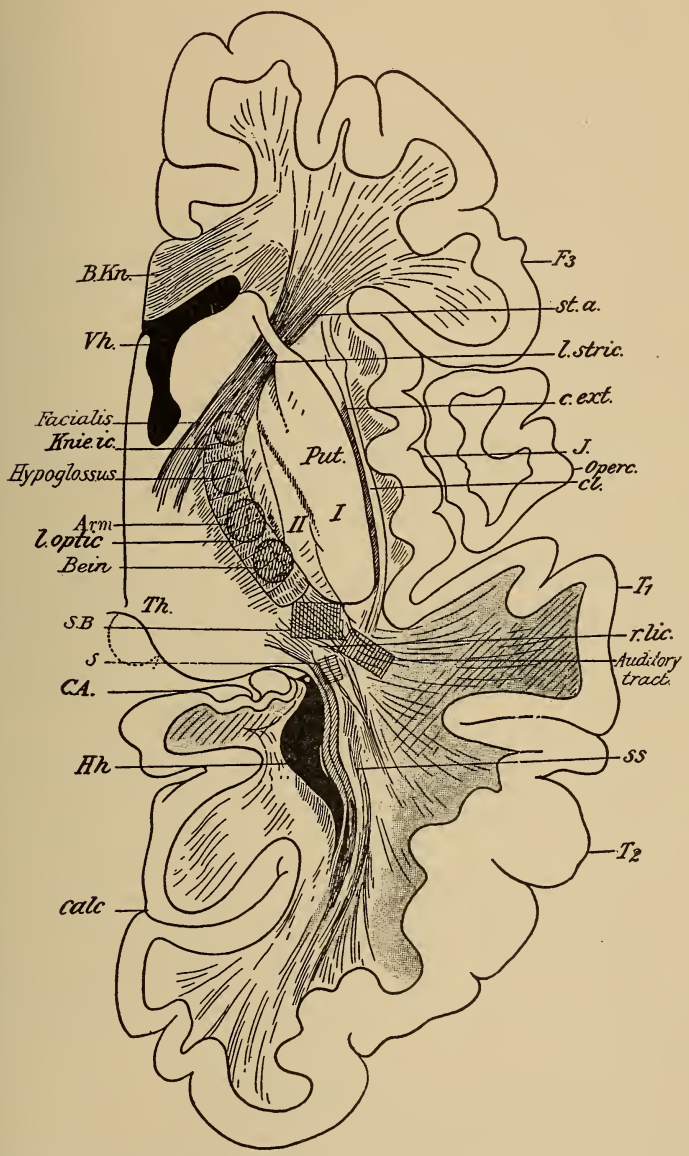
Occipital Lobe.—Irritative lesions of the occipital lobe may cause convulsions with visual warnings. Destructive lesions involving the cuneate lobule cause simple or homonymous hemianopsia; those involving the angular gyrus cause bilateral contraction of the field of vision, greater on the side opposite the tumor, and visual aphasia (word-blindness), especially if situated on the left side.

Temporosphenoidal Lobe.—Irritative lesions of the temporosphenoidal lobe may cause convulsions with auditory warnings or auditory hallucinations. Destructive lesions cause deafness on the opposite side, and sensory aphasia (word-deafness), especially if situated on the left side. Lesions of the uncinate gyrus may cause olfactory and gustatory hallucinations.

Centrum Ovale.—Lesions of the centrum ovale may involve the motor, sensory, or association fibers that pass through it. Subcortical lesions cause motor or sensory disturbances similar to those mentioned in connection with the motor and the sensory cortex, but varying with the situation of the lesion (the fibers implicated). Convulsions do not occur unless the motor cortex is involved; if they do supervene, it is not until the paralysis has been present for some time (the reverse of cortical lesions). Lesions of the centrum ovale lower down resemble those of the internal capsule.

Internal Capsule.—In the comparatively constricted region of the internal capsule practically all of the motor and sensory projection fibers of the cerebral cortex are closely packed together. The internal capsule is made up of a so-called knee; an anterior, lenticulo-caudate (lenticulo-striate) limb; and a posterior limb, of which the anterior two-thirds is called the lenticulo-optic (lenticulo-thalamic) segment, and the posterior one-third the retrolenticular segment. The different sets of neurons passing through the internal capsule are shown in Fig. 20. A lesion of the anterior segment, if not large, may cause only disorders of the higher mental and psychic faculties. A lesion of the knee gives rise to disturbances of the motor nerves of the eye, the motor part of the trigeminus, the facial, and the hypoglossal; in consequence, in addition to the obvious paralyses, disorders of articulation ensue, and if the lesion be on the left side, aphasia. A lesion just posterior to the knee (the pyramidal tract), implicating in order the neurons for the face, arm, and leg, causes usually hemiplegia of the opposite half of the body; monoplegia is very rare, the lesion scarcely ever being small enough to involve

FIG. 20



A horizontal section through the right hemisphere to show especially the internal capsule, the situation of common lesions therein, and their consequences: *B. Kn.*, knee of the corpus callosum; *Vh.*, anterior horn of the lateral ventricle; F_3, inferior part of the third frontal convolution; *l. stric.*, lenticulo-striate division of the internal capsule; *Knie. ic.*, knee of the internal capsule; *l. optic*, lenticulo-optic division of the internal capsule; *Th.*, optic thalamus; *J.*, island of Reil; *cl.*, claustrum; *Operc.*, operculum; T_1, first temporal convolution; *r. lic.*, retrolenticular region of the internal capsule; *C. A.*, ammon's horn; *calc.*, calcarine fissure; *Hh.*, posterior horn of the lateral ventricle; *SS*, optic radiation of Gratiolet; T_2, second temporal convolution; *Facialis*, position in the capsule of the motor tract to the face; *Hypoglossus*, position of the tract to the tongue; *Arm*, position of the tract to the arm; *Bein*, position of the tract to the leg; *S. B.*, sensory fibers; *S.*, visual tract; *Auditory tract*, auditory tract. (After von Monakow.)

only the neurons innervating one limb. A lesion of the posterior third (the retrolenticular segment) of the posterior limb of the internal capsule (which transmits sensory fibers for the entire body, as well as the optic and auditory neurons) gives rise to hemianesthesia (and related sensory changes) of the opposite half of the body, homonymous hemianopsia, and deafness on the opposite side. In most cases, however, notably in cerebral hemorrhage (of which the internal capsule is the common seat), the results are usually varying combinations of those mentioned.

Thalamus.—Lesions of the thalamus usually implicate also the internal capsule, so that the symptoms are likely to be chiefly those just mentioned. In addition, there may be also loss of control of the facial expression and of emotional movements, and perhaps also imperfect control of the posture of the body, athetoid movements of the extremities, etc.

Corpora Quadrigemina.—Lesions (usually a tumor) of the anterior bodies of the corpora quadrigemina cause oculomotor paralysis (pressure on the nuclei or subcortical interruption of impulses), dilated pupils inactive to light and accommodation, and, perhaps, nystagmus. Lesions of the posterior bodies give rise to auditory disturbances, deafness, difficulty in maintaining equilibrium, and a tendency to fall, usually to the opposite side.

Crus; Cerebral Peduncle.—A lesion of one peduncle, as of the internal capsule, causes hemiplegia (face, arm, and leg) and hemianesthesia on the opposite side of the body; paralysis of the ocular motor nerve of the same side (involvement of the nucleus or fibers); perhaps, also, if the lesion (tumor) is large, paralysis of the fourth, fifth, and sixth nerves.

Pons.—A lesion in the pons causes contralateral hemiplegia and hemianesthesia, and usually paralysis of the face (facial nuclei) of the same side (crossed paralysis). In some cases there is also homolateral paralysis of the third, the fourth, the motor division of the fifth, and the sixth nerves; contralateral hemianesthesia (arm and leg) and homolateral anesthesia in the area of distribution of the sensory division of the fifth nerve (crossed hemianesthesia); or homolateral deafness (involvement of the eighth nerve). The lesion (tumor) if situated and growing near the median line of the pons, may cause motor paralysis (sometimes sensory paralysis) of one side; and paralysis of both facial (rarely other cranial) nerves.

Medulla.—A lesion in the medulla may involve one or both pyramidal tracts (which here are close together), and cause, therefore, unilateral or bilateral paralysis (usually more marked on one side than the other); and also paralysis of any or all of the cranial nerves from the eighth to the twelfth (bulbar paralysis, page 850). There may be also disturbances of respiration, disorders of the rate and rhythm of the heart, and glycosuria or polyuria.

Pituitary Body.—Tumors of the pituitary body (akromegaly, page 356) usually compress the optic chiasm and cause at first double temporal hemianopsia (involvement of the fibers that supply the inner half of each

retina, the temporal field of vision). Eventually, with growth of the tumor and involvement of the temporal portions of the optic chiasm and nerves (nasal field of vision), more or less complete blindness ensues.

Cerebellopontine Angle.—Tumors of the cerebellopontine angle cause homolateral disturbances of hearing and of equilibration from involvement of the auditory nerve, which with the tumor seems usually to be intimately related; homolateral paresis or paralysis of the fifth, sixth, and seventh cranial nerves (from compression); controlateral hemiplegia and hemianesthesia from compression of the motor and sensory tracts in the pons; and evidences of disturbance of the cerebellum of the same side.

Cerebellum.—Lesions (tumors) of the cerebellum give rise to homolateral motor weakness and hypotonia of the arm, leg, neck, and trunk; sometimes contralateral hemiplegia (from pressure on the pons and medulla); incoördination, usually more marked on the side of the lesion; inability to maintain equilibrium, an unsteady gait, and a tendency to turn and fall, usually toward the side of the tumor; and nystagmus. Any of the regional cranial nerves may be affected by compression. Vertigo is often very marked, and suggests Ménière's disease. Muscular rigidity, especially of the muscles of the neck, back, and trunk (opisthotonos) may occur, and may be associated with muscular twitchings or tonic spasms, especially in disease of the middle lobe; in this event, the tendency may be to fall backward.

Spinal Cord.—The localization of focal lesions of the spinal cord presupposes a knowledge of the functions of the segments of the cord, of the segmental muscular and sensory innervations, and of the reflex arcs represented in each segment. These are shown in Plate I, page 803, and in the accompanying tables, I, II, and III, arranged by M. A. Starr (pages 838 and 839).

Total Transverse Lesions.—A total transverse lesion, one in which that part of the cord below the segmental level of the lesion is entirely removed from all influences emanating from above, gives rise to: (1) Complete flaccid paralysis of all the muscles innervated by the cord below the segmental level of the lesion (this is in contrast to subtotal transverse lesions in which spastic paralysis occurs); (2) complete anesthesia below the segmental level of the lesion; (3) absent knee-jerks, and other deep reflexes below the segmental level of the lesion (occasionally certain of the cutaneous reflexes, such as the plantar reflex—dorsal flexion of the toe, the Babinski sign—may be preserved); (4) atrophy of the paralyzed muscles; (5) partial or complete reactions of degeneration; and (6) loss of control over the vesical and rectal sphincters.

Subtotal Transverse Lesions.—Subtotal transverse lesions give rise to motor paralyses varying with the segmental level (Table I, page 838), segmental sensory changes shown in Plate I, page 803, and disturbances of reflexes shown in Table II, page 839. In general, the important symptoms are those of myelitis (page 880).

Unilateral Lesions (Brown-Séquard Paralysis).—Lesions limited to one lateral half of the spinal cord are occasionally seen in stab wounds, bullet wounds, syphilis, and tumors, and are rarely observed also in syringomyelia and spinal hemorrhage. They give rise to the following characteristic symptom-complex, named after Brown-

Table I.—*Showing the Muscles Represented in Groups of Cells in the Various Segments of the Spinal Cord.*

II., III. Cervical.	IV. Cervical.	V. Cervical.	VI. Cervical.	VII. Cervical.	VIII. Cervical.	I. Dorsal.
Diaphragm. Sterno-mastoid. Trapezius. Scalenus.	Diaphragm. Lev.ang.scap. Rhomboid. Supra- and infraspin. Deltoid. Supin. long. Biceps.	Rhomboid. Supra- and infraspin. Deltoid. Supin. long. Biceps. Supin. brev. Serratus mag. Pect. (clav.). Teres minor.	Biceps. Serratus mag. Pect. (clav.). Pronators. Triceps. Brach. ant. Long extensors of wrist.	Pronators. Triceps. Brach. ant. Long extensors of wrist and fingers. Pect. (costal). Latis. dorsi. Teres major. Long flexors of wrist and fingers.	Long flexors of wrist and fingers. Extensor of thumb. Intrinsic muscles of hands.	Extensor of thumb. Intrinsic muscles of hands.

I. Lumbar.	II. Lumbar.	III. Lumbar.	IV. Lumbar.	V. Lumbar.
Quadr. lumb. Obliqui. Transversalis. Psoas. Iliacus.	Psoas. Iliacus. Sartorius. Quad. ext. cruris.	Quad. ext. cruris. Obturator. Adductores.	Obturator. Adductores. Glutei.	Glutei. Biceps femoris. Semi-tend. Popliteus.

I. Sacral.	II. Sacral.	III. Sacral.	IV. and V. Sacral.
Biceps femor. Semi-memb. Ext. long. dig. Gastroc. Tibialis post.	Gastroc. Tibialis post. Tibialis anticus. Peronei. Intrinsic muscles of foot.	Peronei. Intrinsic muscles of foot.	Sphincter ani et vesicæ. Perineal muscles.

Séquard: (1) On the side of the lesion: (*a*) motor paralysis on a level with and below the lesion—flaccid paralysis and atrophy, with the reactions of degeneration, of the muscles innervated by the segment of cord destroyed, and spastic paralysis of the muscles innervated by lower motor neurons below the destroyed segment of the cord (due to degeneration of the pyramidal tract—upper motor neuron); (*b*) a segmental zone of complete anesthesia (protopathic, epicritic, and deep sensibility) corresponding to the spinal-cord segment destroyed; and (*c*) below

TABLE II. — *Localization of Muscular Reflex Acts in the Spinal Cord.*

Reflex acts.	Localization in segment.
Pupillary reflex through the sympathetic: Dilatation of the pupil produced by irritation of the neck.	Fourth cervical to first dorsal.
Scapular reflex: Irritation of the skin over the scapula produces contraction of the scapular muscles.	Fifth cervical to first dorsal.
Biceps and supinator longus: Tapping their tendons produces flexion of the forearm.	Fifth and sixth cervical.
Triceps reflex: Tapping tendon produces extension of forearm.	Sixth cervical.
Scapulohumeral reflex: Tapping the inner lower edge of the scapula causes adduction of the arm.	Seventh cervical.
Tapping extensor tendons at the wrist causes extension of the hand.	Sixth to eighth cervical.
Tapping flexor tendons at the wrist causes flexion of the hand.	Seventh to eighth cervical.
Palmar reflex: Stroking palm causes closure of fingers; finger clonus.	Eighth cervical to first dorsal.
Abdominal reflex: Stroking side of abdomen causes retraction.	Ninth to twelfth dorsal.
Genital reflex: Squeezing the testicle causes contraction of the abdominal muscles.	First to third lumbar.
Patella tendon: Striking tendon at knee causes extension of the leg; "knee-jerk."	Second and third lumbar.
Achilles tendon reflex: Tapping the Achilles tendon causes flexion of ankle.	First to third sacral.
Foot clonus: Extension of Achilles tendon causes flexion of the ankle.	First to third sacral.
Plantar reflex: Tickling sole of foot causes flexion of the toes.	First to third sacral.
Babinski's reflex: Scratching sole of foot causes extension of great toe and flexion of the others.	
Mendel's reflex: Tapping the tendons of the toes causes flexion or extension of the toes.	

TABLE III. — *Localization of Skin Reflexes in the Spinal Cord.*

Reflex acts.	Localization in segment.
Epigastric reflex: Stroking breast causes dimpling of the epigastrium.	Seventh to ninth dorsal.
Cremasteric reflex: Stroking inner side of thigh causes retraction of scrotum.	First and second lumbar.
Gluteal reflex: Stroking buttock causes dimpling in the fold.	Fourth to fifth lumbar.

the level of the lesion—normal or increased protopathic and epicritic sensibility, but usually lessening of the deep sensibility (muscle sense). (2) On the side opposite the lesion: (*a*) lessened protopathic (pain) and epicritic (tactile and temperature) sensibility; and (*b*) normal deep sensibility, muscular power, and reflexes.

APHASIA.

The following statements are largely adapted from Starr's discussion of aphasia:

The basis of language is a series of memory pictures of: (1) The sound of a word; (2) the effort necessary to enunciate it; (3) the appearance that its printed or written symbols present; and (4) the effort needed to

Fig. 21

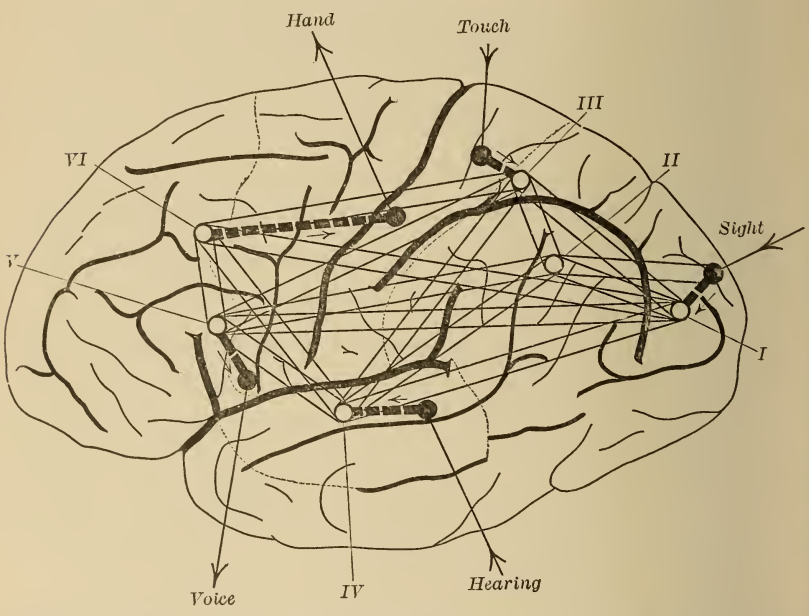


Diagram to illustrate aphasia. The cortical, sensory, and motor centres are indicated by the arrows. The secondary cortical centres of memories are indicated by circles. *I*, visual, of objects; *II*, visual, of words; *III*, tactile; *IV*, auditory; *V*, speech; *VI*, writing. These are joined to one another by association fibers which transmit impulses in both directions. Subcortical lesions in these fibers as well as lesions in the cortex, cause aphasia. (Starr.)

produce these symbols in writing. These memory pictures are intimately connected with one another by means of association fibers which pass in all directions between the areas in which they are located, and they are also joined to the memory pictures which make up the concept (Fig. 21). They are so intimately joined that if one is lost all the

others feel the effect, and a break in the mechanism leads to a defect in the act of speech. The loss of speech or of the power of comprehending the spoken or written language is termed aphasia; the loss of concepts (inability to understand the uses of familiar objects) is termed apraxia.

Etiology.—Aphasia is occasionally said to be functional in nature, being due to a temporary disturbance of the speech centres, such as may occur in epilepsy, hysteria, intoxications like alcoholism, uremia, etc., and angiospasm of the cerebral bloodvessels (in which condition there is usually also some anatomical disorder of the vessels). In the great majority of cases, however, aphasia is due to a destructive anatomical lesion, such as cerebral hemorrhage, softening (embolism or thrombosis,) abscess, or tumor; and the lesion may involve the centres (cortical aphasia) or the tracts (subcortical aphasia).

Types of Aphasia.—Aphasia is broadly divided into sensory and motor. Sensory aphasia consists in a lack of power to recognize or to recall the sound or appearance of words. If the sound alone is lost, the condition is termed word-deafness; if the appearance is lost, the condition is termed word-blindness. Motor aphasia consists in a lack of power to initiate the effort and set in action the motor mechanism needed to pronounce or to write a word. The former is termed motor or ataxic aphasia, the latter motor agraphia; they are usually combined.

Word-deafness.—If the memory of the sound of a word is lost, the word cannot be called to mind and cannot be recognized when heard. If, having been shown a watch or other familiar object, the patient is unable to name it, and does not recognize the correct name when stated, he is totally word-deaf, unable to understand what is said to him. The lesion is in the middle part of the cortex of the first and second temporal convolutions in the left hemisphere in right-handed, and in the right hemisphere in left-handed persons. Since the patient is unable to call to mind the sound of a word, he is unable to say it; but the motor act of pronunciation may be initiated if the impulse can be sent to its centre without passing through the auditory centre. This is the case in exclamations, when one does not choose his words; many patients can swear, although they cannot talk. If the patient exhibits recognition of the name of a familiar object when it has been stated, he has only partial word-deafness, or auditory amnesia. He retains the sound-memory picture (in the cortex), and it can be aroused by hearing it (transmission by the auditory tract to the cortex); but he cannot make the connection between the sight of the object and the sound-memory picture. The lesion, therefore, is subcortical.

Word-blindness (Alexia, Visual Aphasia).—If the memory of the appearance of a word is lost, the usual image of it cannot be called to mind or recognized; the patient is therefore unable to read, because the shapes of the letters and words seen arouse no recollection, and he is unable to write spontaneously, because he cannot remember how the letter looks that he wishes to write. Persons who are word-blind can

sometimes write at dictation or copy, and yet show no evidence of understanding what has been written; the writing centre has been called into activity through some association tract without the intervention of the word-memory picture. A distinction must be made between those who have lost the memory picture (cortical word-blindness) and those in whom it cannot be recalled by ordinary means (subcortical word-blindness). Visual amnesia with word-blindness is due to a lesion involving the inferior parietal convolutions and angular gyrus in the left hemisphere in right-handed persons, and *vice versa*. It may be associated with psychical blindness. Word-deafness and word-blindness frequently occur together; the lesion involves the temporal convolutions and the angular gyrus.

Optical Aphasia, Transcortical Aphasia, or Intercortical Sensory Aphasia.—When the association fibers between the memories of sight and the memories of sound are severed, a condition of aphasia results, which is characterized by an inability to recall the name of a thing seen and to picture to the mind the appearance of a thing named; yet the name is recognized when heard, and the object is recognized when seen. The lesion lies in the long association tract within the temporal and occipital lobes.

Motor Aphasia (Aphemia).—If the memory of the effort needed to pronounce a word is lost, a true paralysis of active speech occurs, although the muscles may not be weakened. The loss of speech involves a loss of the power of repeating words after one another, as well as of voluntary speech, and is not accompanied by any inability to understand spoken or written language. This is the ordinary form of motor aphasia and is due to a lesion of Broca's centre in the posterior part of the left third frontal convolution in right-handed persons.

Agraphia.—Loss of the effort memories necessary for writing is known as agraphia: the pen cannot be used; copying, writing at dictation, and voluntary writing, are all lost. The condition is usually associated with motor aphasia. When a word cannot be recalled to mind, or read, or mentally enunciated, in the majority of persons it cannot be written; but words then can often be written at dictation, if the person is one who has written much. Hence, sensory agraphia and motor agraphia must be distinguished, the former being a part of word-blindness, the latter not at all associated with inability to read. The lesion in motor agraphia is not certainly known, though a few facts point to the posterior part of the second frontal convolution as the probable seat of this function.

Paraphasia, or Intercortical Motor Aphasia; Aphasia of Conduction.—The several memory pictures which are united in the word-image may reasonably be regarded as separate from one another in their location in the brain. But since they are joined together to form the word-image, it follows that the association fibers joining the various areas are as necessary to the use of even a single word as the various areas with their memories; it is really by association only that an object

or a word becomes a subject of thought or use. If these associations are broken, the result is a defect of language, characterized by the misplacement of words, and the patient talks a jargon. There are as many phases of this so-called paraphasia, or intercortical aphasia, as there are association tracts. In intercortical motor aphasia the patient can understand what is heard or seen, and can enunciate words clearly, but is unable to repeat after another person a word heard; he talks rapidly and constantly, trying hard to convey his ideas, but he cannot be understood on account of the mingling of nonsense words or of syllables with words (jargon).

Some lesion of these association fibers is found at the necropsy in almost all cases of aphasia, so that aphasia of purely cortical origin is rare. The lesion of these fibers necessarily causes great confusion in the mental processes of association, so that defects of intelligence are very striking in all cases of aphasia. Marie states that in almost all cases a lesion can be shown in the lenticular zone lying at the bottom of the Sylvian fissure and outside of the lenticular nucleus—the zone through which the majority of these association fibers pass. This he terms the "intellectual centre of language." He regards the motor centre in Broca's convolution as wholly subsidiary, and he considers that its lesion causes anarthria (difficulty in the proper muscular act of speaking without disturbance of intelligence) rather than aphasia. Marie's opinions have not yet been accepted.

The accompanying table (Starr's) shows the chief defects in each variety of aphasia:

TABLE IV.—THE SYMPTOMS IN DIFFERENT FORMS OF APHASIA.

Variety.	Understanding of language.	Power to repeat words.	Power to talk.	Power to read.	Power to write.
1. *Word-deafness.*					
(*a*) cortical,	Lost.	Lost.	Retained (not to answer).	Retained (not aloud).	Retained.
(*b*) subcortical.	Lost.	Lost.	Imperfect (not to answer).	Retained (not aloud).	Retained.
2. *Word-blindness.*					
(*a*) cortical,	Retained.	Retained.	Retained.	Lost.	Lost.
(*b*) subcortical.	Retained.	Retained.	Retained.	Lost.	Imperfect.
3. Intercortical, between T. and O.	Imperfect.	Retained.	Retained.	Imperfect (without comprehension).	Retained (not at dictation).
4. *Motor aphasia.*					
(*a*) cortical,	Retained.	Lost.	Lost.	Retained (not aloud).	Lost.
(*b*) subcortical.	Retained.	Lost.	Lost.	Imperfect (not aloud).	Lost.
5. *Agraphia.*					
(*a*) cortical,	Retained.	Retained.	Retained.	Retained.	Lost.
(*b*) subcortical.	Retained.	Retained.	Retained.	Retained.	Imperfect.
6. Intercortical, between T. and F.	Imperfect.	Lost.	Jargon.	Imperfect.	Lost or imperfect.

APRAXIA, the loss of concepts, includes psychical blindness, psychical deafness, amnesia, and astereognosis.

Psychical blindness is a condition in which there is a loss of visual memories, so that the recognition of objects seen is imperfect and the mind cannot recall the appearance of things formerly familiar; the members of the patient's family, for instance, may not be recognized by sight (optic asymbolia), although they may be identified by the sound of their voices; objects once familiar cannot be named (anomia). The lesion in these cases is in the cortex of the occipital lobe, in the convexity (wide area of the location of visual memory pictures), as well as in the cuneus; commonly in the left hemisphere in right-handed subjects, and *vice versa*. Hemianopsia is usually also present, always when the cuneus is destroyed; and word-blindness (really a part of psychical blindness) may also be associated, although it may occur without psychical blindness.

Psychical deafness (mind deafness) is a condition in which the patient loses the auditory memory of sounds, of musical melodies and harmonies (amusia), and of words (word-deafness), although he may retain the power of hearing; he no longer recognizes sounds or noises, or musical melodies or harmonies once familiar, or words once well known. The lesion is in the left temporal cortex in right-handed persons, and *vice versa*.

Amnesia comprises a destruction of the power of memory—an inability to recall a whole or a part of past experiences. In some cases the power to recall these experiences is only partially lost, lessened, or impeded (delayed), a condition spoken of as hypomnesia; or the condition may be one of perversion of memory (paramnesia).

Astereognosis is a condition in which the patient is unable to recognize objects by the sense of touch (without the aid of sight). The stereognostic sense is a complex made up of tactile, temperature, pressure (weight), and other memories; it is represented in the sensory area of the parietal cortex, especially in the middle third of the posterior central convolution and the adjacent part of the inferior parietal lobule, since stereognostic impressions are in greatest number received by the hands. Astereognosis, therefore, occurs as a symptom of disease of the sensory, cortical, or subcortical area.

Diagnosis.—Starr states that to examine an aphasic subject thoroughly it is necessary to test: (1) The power to recognize objects seen, heard, felt, tasted, or smelled, and their use. This will determine whether the condition of apraxia, or disturbance in the power of recalling any part of a concept, is present. (2) The power to recall the spoken name of objects seen, heard, handled, tasted, or smelled. (3) The power to understand speech and musical tunes. (4) The power to recall to mind objects named. This will test the integrity of the auditory speech area and of the association tracts between other sensory areas and the temporal convolutions. (5) The power to understand printed or written words. (6) The power to read aloud and to understand what is read. (7) The power to recall objects the names of which are seen. (8) The power to write spontaneously and to write

the names of objects seen, heard, etc. (9) The power to copy and to write at dictation. (10) The power to read understandingly what has been written. These tests will determine the condition of the usual word memories in the angular gyrus and of the connections between this area and surrounding sensory and motor areas. (11) The power to speak voluntarily, and, if it is lost, the character of the defects. (12) The power of repeating words after another. This will test the integrity of Broca's centre and its association tracts.

SYSTEM DISEASES OF THE BRAIN AND SPINAL CORD.

Diseases of the Upper (Central) Motor Neuron.

LATERAL SCLEROSIS.

(Spastic Spinal Paralysis, or Paraplegia, of Adults; Spastic Tabes.)

Lateral sclerosis, or spastic spinal paralysis (Erb), is a primary degeneration of the upper motor neuron—that is, of the pyramidal tracts.

Etiology.—The disease is most common in early adult life—after the twentieth year—although an analogous condition may develop in children; and the disorder may be familial. The cause of the disease is not known; it has been attributed to infections, especially syphilis. Doubtless there is a congenital weakness (abiotrophy) of the upper motor neuron, or some primary nutritional change in the motor cells of the cerebral cortex, although some observers doubt whether this ever occurs.

Pathology.—The lesions consist of degeneration of the upper motor neuron, that is, of the pyramidal tracts; this may be confined to the cord or may extend to the cerebral cortex. Following the nerve-fiber degeneration there is a secondary overgrowth of neuroglia.

Symptoms.—The symptoms, which usually develop in, and may be for a very long time, if not entirely, confined to, the legs, consist of a sense of fatigue upon slight exertion, progressive muscular weakness and spasticity, and exaggerated reflexes—knee-jerks, ankle clonus, and the Babinski sign. The rigidity of the muscles occasions a spastic gait, adduction of the thighs (scissors gait), a tendency to stub the toe, etc. There is no muscular atrophy, no sensory disturbance, no ataxia, and no disorder of the sphincters.

Diagnosis.—The diagnosis must be made with circumspection, bearing in mind that primary lateral sclerosis is very rare. Progressive muscular weakness and spasticity, increased reflexes, and the Babinski sign indicate disorder of the lateral columns, but this may be due to disseminated or multiple sclerosis, chronic myelitis, vertebral caries, or spinal tumor and compression, etc.; it may be part of a combined system disease; it may be secondary to a cerebral lesion, such as apoplexy, softening, or tumor; or it may be hysterical. The concomitant symptoms will aid in the diagnosis.

Prognosis.—The disease is slowly progressive, and may continue more than twenty years (Erb).

Treatment.—In the primary cases little benefit can be hoped for, although massage and passive movements may promote the comfort of the patient. Spiller has suggested surgical division of some of the posterior lumbar roots, with a view to lessen the spasticity, and has had the operation done with benefit.

SPASTIC PARALYSIS OF INFANTS.

(Cerebral Palsies of Children; Birth Palsies; Spastic Diplegia, or Paraplegia, or Hemiplegia of Infants; Spastic Cerebral Paraplegia; Little's Disease.)

The spastic paralyses of infants and children comprise several groups of cases: (1) In most cases the disorder dates from birth; the labor, as a rule, was difficult, and forceps perhaps were used. The primary lesion consists of a more or less extensive meningeal hemorrhage (usually venous), which may be limited to the motor area, but may also extend to the frontal lobes (when the intelligence is likely to suffer). Secondary sclerosis and non-development of the brain ensue. (2) In another series of cases there is more or less extensive agenesis of the brain—convolutions, a lobe, or a hemisphere—due to imperfect blood supply (hemorrhage, thrombosis, or embolism). There may be associated porencephalus. (3) In some cases there is a fœtal meningo-encephalitis, which may be syphilitic. (4) Other infants are born prematurely, before the pyramidal tracts have properly developed, and an arrested development is believed to be the cause of the disease (Little's disease). In the different types of cases, syphilis, alcoholism, and neuropathies in the parents are looked upon as etiological factors. Some cases follow postnatal traumatism to the child's head, the infectious diseases, etc.

Symptoms.—The symptoms vary somewhat with the type of the disorder. In the cases due to injury during birth, paralysis may be noticeable during the early days of life, and is likely to be paraplegic (two extremities) or diplegic (four extremities). In other cases the child perhaps may have had convulsions during early life, but noteworthy symptoms are not manifest until the child should begin to walk, when stiffness and inability to use the muscles, especially of the legs, become apparent. In still other cases there may be a sudden onset, attended by unconsciousness and convulsions, and followed by paralysis—which is likely to be hemiplegic, sparing the face. When fully developed there is paralysis—diplegic or paraplegic; the legs are most commonly involved, but the arms also may be affected, and the child may not be able to sit up or support its head (involvement of the neck muscles). The involved muscles are stiff and rigid; the adductor spasm of the thigh muscles causes approximation of the knees or crossing of the legs, and impedes or prevents walking. The reflexes are exaggerated; irregular, spasmodic movements of the muscles are common, especially on volun-

tary movement (spastic chorea or athetosis, usually bilateral); epilepsy is a frequent sequel; the mentality is usually imperfectly developed, and imbecility or idiocy may ensue.

Diagnosis.—The diagnosis is usually apparent from the history of the development of the disease and the mentioned symptoms.

Prognosis.—The outlook is bad, although some improvement in the physical and mental condition may be effected by carefully supervised physical and educational measures.

Treatment.—The treatment of the developed disorder comprises general hygienic measures, massage, electricity, and orthopedic appliances, but they usually avail very little. More attention should be paid to mental training, which often results in notable improvement in the mentality of the children. Since most of the cases result from meningeal hemorrhage during birth, Cushing has advised and practised with good result, opening the skull and removing the blood clot soon after birth.

HEREDITARY SPASTIC SPINAL PARALYSIS.

(*Hereditary Spastic Paraplegia.*)

Hereditary spastic spinal paralysis is a rare disorder, occurring in several members of the same family—in one or more generations. The conspicuous lesions consist of degeneration of the pyramidal tracts throughout the cord, although often most marked in the lower thoracic and the lumbar regions. The nature of the disorder is not known; it is assumed to be an abiotrophy—imperfect development and deficient potentiality of the upper motor neuron, in consequence of which it succumbs in early life. The *symptoms* consist of the development during infancy, childhood, or adolescence of muscular rigidity and paralysis, involving especially the legs; and increased reflexes, including the Babinski sign. There are no sensory disturbances and no disorders of mentality. The disorder is usually slowly progressive, and is not amenable to any treatment.

AMAUROTIC FAMILY IDIOCY.

(*Hereditary Spastic Paraplegia—Cerebral Type; Tay-Sachs' Disease.*)

Amaurotic family idiocy is described by Sachs as a family disease of infancy of unknown etiology, and of which the basis is "a nervous system so inadequate to the demands imposed upon it that its cells, after having performed their function for a few weeks or months undergo complete disintegration." The condition, therefore, is also an abiotrophy, and is akin to hereditary spastic spinal paralysis; both disorders are often described as, respectively, the spinal and the cerebral type of hereditary spastic paraplegia. The chief *symptoms* of the cerebral type, or amaurotic family idiocy, are thus summarized by Sachs: Mental impairment

during the first months of life, leading to absolute idiocy; paresis, or paralysis of the greater part of the body, which may be flaccid or spastic; normal, deficient, or increased reflexes; diminution of vision, terminating in absolute blindness (the cherry-red spot in the region of the macula lutea, and later a simple optic atrophy); marasmus, and a fatal termination, as a rule, before the age of two years; the occurrence of the affection in several members of the same family; and in some, but not all, cases, nystagmus, strabismus, hyperacusis, and convulsions. Since the disease has been frequently observed in the children of blood relations, Sachs advises discouraging consanguineous marriages, with a view to prevent the birth of such amaurotic children.

UNILATERAL ASCENDING AND UNILATERAL DESCENDING PARALYSIS.

Unilateral ascending and unilateral descending paralyses comprise symptom-complexes, first described by Mills, in which unilateral primary degeneration of the pyramidal tract (upper motor neuron) occurs, and of which the clinical expression is unilateral spastic paralysis, beginning as a rule, in the lower extremity and ascending. Mills states that the disorder may be a primary degeneration of the pyramidal tracts, or may occur in focal disease (tumor or softening) of the brain or cord, in multiple sclerosis, in cerebrospinal syphilis, in hysteria, and as unilatera amyotrophic lateral sclerosis or paralysis agitans.

Diseases of the Lower (Peripheral) Motor Neuron.

CHRONIC ANTERIOR POLIOMYELITIS.

(Progressive Spinal Muscular Atrophy; Progressive Muscular Atrophy of the Duchenne-Aran and the Vulpian Type.)

Etiology.—Chronic anterior poliomyelitis, or progressive spinal muscular atrophy, is most common in middle life (excluding the infantile cases). It may develop upon an obsolete acute poliomyelitis (page 270), or may be due to syphilis, lead poisoning, etc.

Pathology.—The lesions consist of degeneration and atrophy of the motor nuclei in the anterior cornua of the spinal cord, and corresponding atrophy of the motor roots. In some cases there are associated degenerative changes in the pyramidal tracts (amyotrophic lateral sclerosis).

Symptoms.—The chief symptoms consist of muscular atrophy and proportionate weakness and flaccidity (flaccid paralysis), which are usually first manifest in the small muscles of one or both hands (Duchenne-Aran type), less commonly in the muscles of the shoulder girdle (Vulpian type), and rarely in the muscles of the feet or the trunk There are also diminished or lost reflexes, fibrillary twitchings of the affected

muscles, and the reactions of degeneration (partial or complete). The atrophy is progressive, and ultimately leads to the development of the claw-like hand (main-en-griffe); beginning in the hands, it may later implicate the muscles of the forearm, arm, shoulder, and trunk. There are no sensory changes nor involvement of the sphincters. Occasionally exacerbations of the process are observed (subacute poliomyelitis).

Werding and Hoffman have described a similar disorder, beginning in infancy or childhood, often hereditary, and affecting several members of the same family.

Diagnosis.—The disease should be distinguished from multiple neuritis (pain and tenderness of the nerve trunks and sensory changes), amyotrophic lateral sclerosis (exaggerated tendon reflexes), progressive neural muscular atrophy, the muscular dystrophies, syringomyelia, etc. Some neurologists distinguish between chronic anterior poliomyelitis and progressive spinal muscular atrophy, the chief distinction being the absence of periods of exacerbation in progressive spinal muscular atrophy.

Prognosis.—The disorder is usually progressive, but arrest sometimes occurs.

Treatment.—Massage, electricity, passive movements, tonics, etc., are sometimes useful, but commonly avail little.

PROGRESSIVE NEURAL MUSCULAR ATROPHY.

(*Progressive Muscular Atrophy of the Charcot-Marie-Tooth Type; Peroneal Type of Progressive Muscular Atrophy.*)

Etiology.—Progressive neural muscular atrophy is more common in males than females; it is often an hereditary or family disease, and commonly develops before the twentieth year. No cause is known.

Pathology.—The lesions consist of degeneration of the axons and connective-tissue overgrowth of the peripheral nerves, atrophy of the cells of the anterior cornua of the spinal cord, degeneration of the posterior roots and columns, slight degeneration of the pyramidal tracts, and atrophy of the muscle fibers, with connective-tissue overgrowth.

Symptoms.—The chief symptoms consist of progressive muscular atrophy, weakness, and flaccidity (flaccid paralysis), beginning in the small muscles of the feet, and later involving the peroneal muscles, so that foot-drop ensues. In the course of time the muscles of the thigh may become involved, as well as the small muscles of the hands, and perhaps the forearm. The muscles of the upper arm, neck, trunk, and face are rarely implicated. Associated with the paralyses there are fibrillary twitchings of the affected muscles, diminished or lost reflexes, reactions of degeneration (partial or complete), and often paresthesia, lessened sensibility, and slight pain in the affected peripheral regions.

Diagnosis.—The hereditary or family occurrence of the disease, its development in early life, the almost if not quite complete limitation of the atrophic and other changes to the periphery, and the prominence

of the motor as contrasted with the sensory phenomena, are characteristic of the disease and distinguish it from progressive spinal muscular atrophy (chronic anterior poliomyelitis), multiple neuritis, and the muscular dystrophies.

Prognosis.—The disease is slowly progressive, but, having reached its height, it may remain stationary for years.

Treatment.—No known treatment is of any noteworthy avail, but massage, electricity, passive movements, and orthopedic appliances may afford symptomatic relief.

BULBAR PARALYSIS.

(Inferior Polioencephalitis.)

Bulbar palsy is a paralysis of the motor nuclei of the bulb (pons and medulla)—that is, the motor trigeminal, the facial, the glossopharyngeal, the vagus, the spinal accessory, and the hypoglossal nuclei.

Acute bulbar palsy (inferior polioencephalitis) may be due to acute inflammation (akin to acute anterior poliomyelitis), or to embolism, thrombosis, or hemorrhage of the vessels supplying the bulbar nuclei. The lesions are analogous to those of acute poliomyelitis (page 270), or to those of embolism, thrombosis, or hemorrhage occurring elsewhere in the brain. The onset is usually sudden, and may be attended by headache, vertigo, and vomiting. If death does not soon ensue (which is common, on account of respiratory and cardiac failure) the muscles of the lower part of the face, the lips, the tongue, the palate, and the pharynx are found to be paralyzed. Death may eventually ensue. In some cases there is associated ophthalmoplegia (superior polioencephalitis) or acute poliomyelitis. The cases due to hemorrhage or softening (embolism or thrombosis) may sometimes be distinguished from those due to infection or toxemia (inflammation) by their being more sudden in onset, and usually attended by motor (paralytic) and sensory phenomena due to implication of the related tracts passing through the pons and medulla; and sometimes by the absence of fever and other signs of infection.

Chronic bulbar palsy (glosso-labio-laryngeal and pharyngeal paralysis) occurs especially after the fortieth year, and is of unknown etiology; it is probably an abiotrophy. The lesions consist of bilateral degeneration of the motor nuclei of the bulb, especially the vagus, spinal accessory, and hypoglossal.

Symptoms.—The symptoms consist of the gradual onset and progression of difficulty in articulating (hypoglossal involvement), which is especially marked in connection with labials, such as b, p, and v, and linguals, such as l, m, r, and t. Soon there is difficulty in swallowing (vagus and spinal accessory involvement), and fluids regurgitate through the nose (paralysis of the palatal and pharyngeal muscles); food may pass into the larynx (loss of the pharyngeal reflex), and the

voice takes on a nasal quality and becomes weak. In the course of time the muscles of the lips (orbicularis oris, supplied by the hypoglossal), lower part of the face (facial nerve), and tongue become paralyzed and atrophied, and exhibit fibrillary twitchings and the reactions of degeneration; the tongue cannot be protruded; the lips cannot be puckered (as in whistling); the saliva drools from the mouth; the palatal, pharyngeal, and laryngeal reflexes lessen and finally disappear; and in the course of several years the patient dies from respiratory or cardiac failure, if he has not earlier succumbed to an intercurrent infection. Intelligence is preserved throughout the course of the disease and there are no sensory changes. In some cases there is an associated ophthalmoplegia.

Diagnosis.—The diagnosis is rarely difficult. Some confusion may occur with *pseudobulbar palsy*, a disorder caused by supranuclear (cerebral) lesions; but in this there is usually a history of apoplectiform attacks, the lesions are unilateral or asymmetrical in distribution and non-progressive (in the absence of succeeding apoplectiform attacks), and muscular atrophy, fibrillary twitchings, and the reactions of degeneration do not occur. So-called *asthenic bulbar paralysis*, or *myasthenia gravis* (a disorder of unknown nature, although probably toxic, and muscular rather than nervous in origin), may be distinguished by the rapid exhaustion of the muscles upon slight exertion, and their recovery upon resting; by involvement of muscles other than those innervated by the bulb (such as the limbs and the ocular muscles), and the absence of atrophy and fibrillary twitchings of the affected muscles; and by the presence of the myasthenic reaction (early exhaustion of the muscle, especially when stimulated by the faradic current, and recovery after resting). Postdiphtheritic and other forms of multiple neuritis and the cases due to tumor, meningitis, etc., should be readily distinguished.

Prognosis.—The disease, as a rule, is steadily progressive.

Treatment.—There is no satisfactory treatment. Benefit often attends general hygienic measures, electricity, and strychnine. Special care must be taken in feeding the patient to prevent aspiration of food and consecutive bronchopneumonia.

OPHTHALMOPLEGIA.

(*Superior Polioencephalitis.*)

Ophthalmoplegia, or paralysis of the motor nuclei of the eyeball, is a disorder in every way allied to bulbar palsy, with which it is often associated; like it, it may be acute or chronic, and the etiological factors are analogous. The acute cases may be sudden in onset, attended by headache, vertigo, and vomiting, and are characterized by rapidly developing paralysis of the ocular muscles—except, in some cases, the elevator of the upper lid and the sphincter of the iris. In some cases death ensues in stupor or coma; in other cases the patient may recover (usually neuritic cases), or chronic ophthalmoplegia may ensue. This may also be

a chronic process from the beginning, and is characterized by gradually progressing paralysis of the ocular muscles, commonly bilateral. The paralysis may be partial or complete and may involve one or all the muscles. Diplopia is the chief complaint, but it may disappear in advanced cases. Ptosis may or may not be present. In some cases only the extraocular muscles are involved (external ophthalmoplegia); in some cases only the internal muscles (internal ophthalmoplegia); in other cases, both. The disorder is usually found in association with syphilis, tabes dorsalis, paretic dementia, and progressive muscular atrophy. The treatment is unsatisfactory, but the salicylates seem to be of value in some acute cases, and the iodides in some of the chronic cases, even in the absence of a history of syphilis; in the event of syphilis the iodides should be combined with mercury. The chronic cases are occasionally benefited by electricity and strychnine; and the diplopia may be corrected by glasses.

Disease of the Upper (Central) and the Lower (Peripheral) Motor Neurons.

AMYOTROPHIC LATERAL SCLEROSIS.

(*Progressive Muscular Atrophy; Charcot's Disease.*)

Etiology.—Amyotrophic lateral sclerosis is a disease of adult life and is somewhat more common in men than in women. It is doubtless an abiotrophy, but has been attributed to intoxications like lead poisoning, and to infections, such as syphilis, etc.; but a definite causal relationship has not been shown.

Pathology.—The lesions consist of degeneration and atrophy of the upper and the lower motor neurons—that is, of the pyramidal tracts, the nerve cells of the anterior cornua of the spinal cord, the anterior nerve roots, the peripheral nerves, and the muscles. The degeneration of the pyramidal tracts sometimes extends to the cerebral cortex, involving both the axons and the nerve cells.

Symptoms.—The symptoms consist of a combination of those of lateral sclerosis (weakness and spasticity of the muscles of the legs, with increased knee-jerks, ankle clonus, and the Babinski sign), and of progressive muscular atrophy, beginning in the small muscles of the hands, and associated with fibrillary twitching and partial or complete reactions of degeneration. In the beginning the symptoms in the legs or in the arms may be in the ascendancy, but the atrophy in the course of time extends to the forearm and the upper arm and may involve the muscles of the trunk (with ensuing kyphosis or lordosis) and the thigh; those of the lower leg are usually relatively uninvolved. Sensory phenomena are in abeyance, although there may be slight pain. Rarely the atrophic and other changes begin in the muscles of the shoulder girdle,

or in those supplied by the motor nuclei of the bulb (a type of glosso-labio-laryngeal and pharyngeal paralysis).

Diagnosis.—The diagnosis is to be based upon the co-existence of the signs of lateral sclerosis and of progressive muscular atrophy (involvement of the cells of the anterior cornua of the spinal cord).

Prognosis.—The disease is slowly progressive and commonly terminates fatally within one to three or four years.

Treatment.—Treatment is not of much avail. Symptomatic relief may follow the use of electricity, massage, passive movements, strychnine, etc.

Diseases of the Sensory Neuron.

TABES DORSALIS.

(Locomotor Ataxia; Posterior Sclerosis.)

Etiology.—Tabes dorsalis is almost ten times as common in men as in women; it develops usually during the fourth decade of life, but cases are occasionally encountered in very young subjects (second decade—juvenile cases due to hereditary syphilis), and even about or after the sixtieth year. In about 95 per cent. of the cases (Erb) tabes dorsalis follows syphilitic infection, developing, as a rule, in from five to twenty years after the initial lesion; rarely, it ensues within a year or two. Trauma, exposure to wet and cold, infections other than syphilis, extreme bodily and mental exertion, sexual excesses, etc., are sometimes said to be of etiological significance.

Pathology.—Tabes dorsalis is a disease of the sensory neuron: of the dorsal-root fibers (exogenous fibers), and not of the fibers that run from one part of the cord to another (endogenous fibers); following primary degeneration, secondary sclerosis occurs. The situation of the primary lesion has not been positively determined. Obersteiner and Redlich have accentuated the importance of inflammation of the dorsal pia-arachnoid with involvement of the root fibers, which, as they pierce the pia-arachnoid, are poor in myelin and have no neurilemma; Nageotte believes the process to originate in a meningoneuritis of the dorsal roots centrally to the dorsal ganglia, where they are still covered by the dura mater; Starr states that the primary lesion is in the posterior spinal ganglia and the ganglia of the cranial nerves. In the spinal cord the degenerative and sclerotic lesions are found in the pathway of the dorsal-root or exogenous fibers. Most of these enter directly into the postero-external column (Burdach); some pass into the gray matter; others later run in the postero-internal column (Goll), on account of the superior fibers crowding toward the median line those fibres that have entered lower down; and they end in the nuclei in the dorsal columns of the medulla. Other of these root fibers enter directly into a small bundle of fibers (Lissauer's tract) at the apex of the posterior cornua and ascending or descending a short distance, enter the cornua,

where some of them end; others pass forward to the cells of the anterior cornua or cross over to the opposite side and enter the anterolateral column. Involving these pathways, the sclerosis usually begins, or is most marked, in the lumbar region and commonly decreases from below upward; rarely it may begin in the sacral, thoracic, cervical, or bulbar regions. Changes are often found in the cranial nerves and nuclei, especially the optic and ocular nerves, the pneumogastric, etc.; in the motor tracts and anterior horns of the spinal cord (this accounting for the muscular atrophy of some cases); and in the cerebral cortex (tabo-paralysis; paretic dementia).

The lesions are commonly described as parasyphilitic rather than truly syphilitic, but the radicular meningitis often found and the ready demonstration of the Wassermann reaction indicate the essentially syphilitic nature of the process.

Symptoms.—The symptoms are quite variable in their mode of development and in their mutual association. They can perhaps be best comprehended by bearing in mind: (1) That they result from dorsal-root lesions, and that a dorsal root conveys fibers for epicritic, protopathic, and deep sensibility (page 803), as well as fibers composing part of a reflex arc; (2) that the disease may begin at varying levels of the cord; and (3) that not all the fibers in a dorsal root are diseased at once or to the same extent. The disease is sometimes divided clinically into the first, incipient, painful, or pre-ataxic stage, the second or ataxic stage, and the third or paralytic stage. In practice these are not always separable the one from the other.

The chief symptoms of the early stage of the disease consist of: Sharp shooting, fulgurant, or lancinating pains, usually in the legs, rarely in the arms (cervical tabes); loss of the deep reflexes, especially the knee-jerk (Westphal's sign), and of the Achilles tendon reflex; inequality of the pupils and the Argyll Robertson pupil (immobility to light, but contraction during convergence and accommodation); and swaying of the body, perhaps progressing to falling when the eyes are closed (Romberg's sign). In some cases complaint is of dull aching, rather than of sharp pains, and these being interpreted as "rheumatic," the disease is for a long time overlooked. Other patients complain early of various paresthetic phenomena—such as numbness or tingling of the feet, a sensation as if walking on cotton or eggs, etc.; or of delay or difficulty in emptying the bladder; or of loss of sexual power and desire; or of transient or permanent diplopia (involvement of the cranial nerves); or of gradually increasing dimness of vision (primary optic atrophy); or of so-called visceral crises, such as gastric crises (severe epigastric pains and vomiting), laryngeal crises (dyspnœa and stridor), renal crises (simulating renal colic), rectal crises, etc. These symptoms, if they do not occur early, are likely to come on later, and in varying combinations. Sensory disturbances, as a rule, are readily found early if searched for, and commonly consist of segmental hyperthesia or anesthesia, often about the chest or abdomen or in the legs and soles of the feet.

In the course of time ataxia supervenes: the patient at first experiences difficulty in walking in the dark; the Romberg sign will be found upon examination, but soon the characteristic ataxic gait supervenes—the foot being considerably elevated, thrown out with an ill-controlled jerk and brought down with a thud, and an effort being made to control these movements by close attention with the eyes. Ataxia may also supervene in the arms. About this time hypotonia of the muscles may be observed, although it often precedes the ataxia; it consists of abnormal flaccidity of the muscles (and sometimes also of the ligaments), so that passive movements of the limbs much beyond the normal range are possible, although there may be no loss of muscular power. There are varied sensory disturbances: the pains and paresthetic phenomena may continue; bands of segmental hyperesthesia or anesthesia are common; the transmission of painful stimuli and the response thereto are delayed; and the deep sensibility, especially the sense of location, becomes impaired. Trophic manifestations also ensue, especially herpes zoster, a perforating (usually painless) ulcer of the sole of the foot near the metatarsophalangeal joint of the great toe, and Charcot's arthropathies—painless swelling of the large joints, especially the knee (due to increase of fluid, enlargement and exostosis of the ends of the bones, and atrophy and absorption of the bone and the articulating cartilage). The bones may become brittle and readily fractured.

After some years, if the patient does not sooner die, the third, or paralytic stage, develops: the patient, in addition to being ataxic, becomes muscularly weak, unable to walk, and finally bedridden.

In a certain proportion of the cases tabes becomes associated with *general paralysis of the insane* (general paresis, paralytic dementia), giving rise to a condition sometimes spoken of as *tabo-paralysis*. Some writers maintain the essential etiological (syphilitic) and pathogenetic identity of the two disorders, and believe that the one or the other may be the antecedent or the secondary disorder in a given case. The majority of cases of tabes dorsalis, nevertheless, are devoid of noteworthy mental symptoms, and general paretic subjects, as a rule, are not tabetic. However, in some cases of tabes, after the lapse of time, evidences of cerebral disorder (chronic meningo-encephalitis) supervene: the subject becomes irritable; his character changes, he becomes intemperate, licentious, and neglectful of himself, his family, and his business; and his memory fails. Soon mental deterioration becomes more marked, the general condition being that of exaltation and well-being and delusions of grandeur, or there may be alternating periods of exaltation and of depression. In addition there are disturbances of speech, which becomes slowed; there is special difficulty in pronouncing labials and linguals; tremors of the lips, tongue, face, and hands (especially obvious in attempting to write) become noticeable; and the patient gradually develops a general muscular weakness, although the mental disorder may lead him to deny any bodily infirmity. Transient paralyses, involving the cranial nerves, or hemiplegia, attacks of stupor, coma, or

momentary loss of consciousness, convulsive seizures, especially of the face or arm, may supervene. Finally the dementia becomes aggravated, the patient takes permanently to his bed, and ultimately dies. The significant symptoms of tabes are variously associated with these manifestations of the cerebral disorder.

Diagnosis.—The diagnosis of tabes dorsalis should be readily made by attention to the aforementioned symptoms. Efforts should be made to recognize the disease in its earliest stage; the presence of any two of the following signs is of the greatest diagnostic importance: lightning pains, Argyll Robertson pupil, lost knee-jerks and other deep reflexes, and Romberg's sign; and any one of these in association with two of the other mentioned symptoms warrant the diagnosis. In doubtful cases a history of syphilitic infection, a positive Wassermann reaction, or an excess of lymphocytes in the cerebrospinal fluid, will furnish material diagnostic aid. Multiple neuritis should be readily excluded by the absence of motor paralysis, muscular atrophy, tender nerve trunks, etc.

Prognosis.—The disease may pursue a slow or a comparatively rapip course, and, having made more or less progress, it may come to a standstill. Slow progression in the past is a promise of slow progress in the future. The existence of lightning pains for years prior to the loss of knee-jerks augurs slow progression or standstill, and the cases with early optic atrophy are likely also to show delayed onset of ataxia and slow progression of the other symptoms. Degeneration of the columns in the spinal cord, once effected, of course, cannot be remedied.

Treatment.—If the patient is in good physical condition and the disease is not far advanced, a course of efficient antisyphilitic treatment should be instituted, especially if the infection were poorly, or not all, treated. Otherwise, attention should be directed to improving the general nutrition of the patient by good food, rest, and tonics, such as iron, quinine, and strychnine, and hydropathic measures, massage and electricity. Arsenic and silver nitrate have been recommended. The lightning pains may be benefited by aspirin, acetphenetidin, and other coal-tar derivatives, aluminum chloride, codeine, and morphine (which are almost certain to engender the habit). The ataxia may be markedly improved by exercises—which have been systematized for the tabetic subject by Fraenkel.

INTERSTITIAL HYPERTROPHIC PROGRESSIVE NEURITIS OF CHILDHOOD.

Interstitial hypertrophic progressive neuritis of childhood is a hereditary or familial disorder, studied especially by Dejerine, that develops in childhood or adolescence from unknown causes, although it has been attributed to syphilis. The chief lesions consist of degeneration of the posterior roots and columns of the cord (as in tabes dorsalis) and of parenchymatous and interstitial changes in the peripheral nerves,

especially of the legs. The *symptoms* consist of those of tabes, especially ataxia of the arms and legs, severe pains, sensory disturbances (as in tabes, with retardation of the appreciation of painful stimuli), inequality of the pupils, Argyll Robertson's sign, etc.; general muscular atrophy, especially marked at the periphery; and kyphoskoliosis (due to weakness of the trunk muscles). The palpable nerve trunks are thickened and indurated. There is no satisfactory treatment.

Combined System Diseases.

HEREDITARY OR FAMILY ATAXIA.

(Friedreich's Ataxia.)

Etiology.—Friedreich's ataxia is a family disease, that may or may not have occurred in the antecedents. It is slightly more common in males than in females, and is likely to develop in early life—often in childhood, and in the great majority of cases before puberty. The cause is not known. Alcoholism and syphilis in the parents seem to be inoperative. Evidently the disease is an abiotrophy.

Pathology.—The lesions consist of degeneration and consequent sclerosis of the posterior, lateral, and direct cerebellar tracts, and sometimes also of Gowers' anterolateral tracts.

Symptoms.—The symptoms consist of the gradual development during early life of ataxia. This occurs first in the legs, and gives rise to a swaying or stumbling gait, which usually becomes aggravated on closing the eyes (Romberg's sign); developing later in the arms, it occasions irregular choreiform movements. The head also may be subject to similar movements; and irregular, oscillatory movements may affect the body when it is erect at rest, or a limb when it is extended (static ataxia). Nystagmus and a slow scanning or stumbling speech often supervene. The deep reflexes are lost; the skin reflexes are usually preserved; there are no noteworthy sensory changes. In advanced cases, on account of muscular weakness and atrophy, talipes equinus, hammer-toes, or spinal curvature may occur.

Diagnosis.—The diagnosis should be quite apparent from the onset of the aforementioned symptoms in early life, especially if several members of a family are affected .

Prognosis.—The disorder is slowly progressive.

Treatment.—No satisfactory treatment is known; the available treatment is purely symptomatic.

HEREDITARY OR FAMILY CEREBELLAR ATAXIA.

(Marie's Ataxia; Hereditary or Family Spastic Paraplegia.)

Hereditary or family cerebellar ataxia is a family disorder, that tends to develop after puberty or in early adult life (the third decade), and is characterized anatomically by gross atrophy of the cerebellum or by

microscopic atrophy of the cells of Purkinje. Clinically, the disorder resembles Friedreich's ataxia in the family occurrence, the ataxia (although the gait is likely to be less awkward), the Romberg's sign, the choreiform movements, the nystagmus, and the disturbances of speech; but it differs from Friedreich's ataxia in its onset later in life, normal or exaggerated knee-jerks, ultimate spasticity of the legs (attributable to degeneration of the pyramidal tracts), and the absence of deformities of the feet and trunk.

ATAXIC PARAPLEGIA.

(Posterolateral Sclerosis.)

Etiology.—Ataxic paraplegia occurs most commonly in males in the fourth and fifth decades. No cause can be assigned; it is not due to syphilis; it has been attributed to cold and wet, traumatism, etc.; it is probably an abiotrophy.

Pathology.—The lesions consist of degeneration and consequent sclerosis of the posterior and the lateral columns; that of the posterior columns differs from that of tabes in being equally marked in the lumbar and thoracic regions and in sparing the dorsal-root zones; that of the lateral columns is most marked in the pyramidal tracts, but may also involve the direct cerebellar and the direct pyramidal tracts.

Symptoms.—The symptoms consist of gradually developing weariness, muscular weakness, and difficulty and unsteadiness in walking. Examination reveals well-marked incoördination (as in tabes dorsalis), which may extend to the arms; and Romberg's sign. The deep reflexes are exaggerated, including ankle clonus and the Babinski sign. In the course of time rigidity and spasticity of the muscles of the legs ensue (spastic paraplegia), and may somewhat overshadow the ataxic phenomena. Sensory and ocular symptoms are rare. Late, the sphincters may become involved, and mental symptoms, suggesting paretic dementia, may develop.

Diagnosis.—The disease should be readily recognized from the ataxic gait, the increased knee-jerks, and muscular rigidity, and the absence of the lightning pains, sensory changes, and ocular signs of tabes.

Prognosis.—The disease is slowly progressive, as a rule, but sometimes arrest occurs.

Treatment.—The treatment is that of primary lateral sclerosis (page 845), plus Fraenkel's method for the reëducation of muscles, useful in tabes dorsalis.

TOXIC COMBINED SCLEROSIS.

(Subacute Combined Sclerosis of the Spinal Cord, Putnam; Diffuse Secondary Degeneration of the Spinal Cord; Anemic Degeneration of the Spinal Cord.)

Etiology.—The disorder, or disorders, comprised within the term toxic combined sclerosis of the spinal cord occur secondarily in the course of toxic and anemic conditions, such as pernicious anemia, severe secondary

anemias, cachectic conditions generally, acute and chronic infections, prolonged suppuration, lead poisoning, ergotism, pellagra, etc.

Pathology.—The lesions consist of degeneration and sclerosis of the posterior columns, usually most marked in the thoracic and cervical regions; and sometimes also of the crossed pyramidal tracts; rarely of other tracts. The degeneration is sometimes focal rather than systemic.

Symptoms.—The symptoms consist of early numbness and tingling of the feet and legs and mild spastic or ataxic paraplegia. Similar phenomena may develop in the arms, and those in the legs become aggravated, so that the patient then exhibits the phenomena of well-marked spastic paraplegia, with diminution or loss of all forms of sensibility in the legs and perhaps the lower part of the trunk. Finally, a stage of flaccid paralysis may supervene.

Diagnosis.—The development of the mentioned symptoms in the course of any of the diseases that may occasion them should serve for the diagnosis.

Prognosis.—The outlook depends more upon the primary disorder than upon the nervous manifestations. As a rule, a fatal outcome supervenes within a few months.

Treatment.—The treatment is wholly that of the primary disorder.

NON-SYSTEM DISEASES OF THE BRAIN AND SPINAL CORD.

MULTIPLE SCLEROSIS.

(*Disseminated Sclerosis; Insular Sclerosis.*)

Multiple or disseminated sclerosis is a disorder characterized by small islets or foci of sclerotic neuroglial tissue scattered irregularly throughout the brain and spinal cord.

Etiology.—Multiple sclerosis occurs especially in early adult life (before the thirtieth year); it affects the sexes about equally. The exciting cause is not known. Some cases are believed to be congenital—an abiotrophy; others appear to follow some of the infectious diseases (typhoid fever, scarlet fever, smallpox, pneumonia, etc.), and still others are attributed to metallic poisons (lead, mercury, etc.).

Pathology.—The lesions consist of foci or islets of sclerotic neuroglial tissue scattered throughout the brain and spinal cord, and sometimes also in the peripheral (especially the cranial) nerves. They are much more common in the white than in the gray matter, and they vary in size from those microscopic to those 3 to 5 mm. or more in diameter. The primary lesion seems to be a neuroglial proliferation (perhaps toxic in origin), with consecutive destruction of the medullary sheaths of the neurons. The lesions are not systemic (that is, not confined to definite tracts); and sparing, at least for a very long time, the axons, they do not give rise to secondary degenerations of tracts or fibers.

Symptoms.—The onset of the symptoms is insidious and their progress slow, and they vary extraordinarily with the seat of the lesions. In the

so-called typical cases the early complaint may be stiffness and weakness of one or both legs, rarely of an arm. Gradually spastic paralysis of the legs develops and is associated with increased knee-jerks, ankle clonus, and the Babinski sign; the deep reflexes of the arm also are usually increased, but the superficial reflexes, especially of the abdomen and the cremasteric reflex, may be abolished. In most cases there is more or less ataxia and a staggering gait (involvement of the posterior and cerebellar tracts). Muscular tremors develop—usually first and most markedly in the arms, but they may involve the legs and the head. They are characterized by being absent or ceasing during rest, by developing upon voluntary motion of any kind, such as writing, taking hold of an object (so-called volitional or intention tremor), by their general coarseness, and by increasing in amplitude, jerkiness, and irregularity upon continuation of the voluntary movement. Nystagmus is common and may develop spontaneously or only upon voluntary movement of the eyes; it may be associated with weakness of one or more ocular muscles and diplopia. Optic atrophy is often present and is rather commonly confined to the temporal half of one or both disks. The pupils usually react normally to light and in accommodation. There is, in most cases, more or less disorder of speech, which is commonly slow, scanning, staccato, syllabic, and in a monotone. Mental impairment ensues to a greater or less degree, and the patient becomes emotionally unstable, laughing or crying upon little or no provocation.

There are a number of other symptoms that occur variously in different cases, such as vertigo, apoplectiform or epileptiform attacks, paresthesias, patchy anesthesias, disorder of the vesical and rectal sphincters, etc. The disease is often characterized by notable improvement or disappearance of some of the obtrusive symptoms and by relapse or recurrence.

Diagnosis.—A combination of the symptoms mentioned is quite characteristic. In the early and ill-developed cases (especially in young women) care must be taken not to confound the disease with hysteria; in hysteria, however, intention tremors, nystagmus, optic atrophy, scanning speech, and the Babinski sign are absent, and various hysterical stigmas are likely to be present. Cerebrospinal syphilis may simulate multiple sclerosis, but it is likely to exhibit pupillary and other phenomena, and rarely, if ever, shows intention tremors, nystagmus, and scanning speech. Paralysis agitans also is characterized by a tremor, but this occurs while the parts are at rest and is fine, nystagmus and scanning speech are absent, and the disease occurs in advanced life. Friedreich's and cerebellar ataxia should be readily excluded.

Prognosis.—The disease is of slow progress; remissions are quite common; standstill may ensue, but recovery is not likely.

Treatment.—The treatment is symptomatic: rest, the avoidance so far as possible of muscular movements (talking, walking, exercise, etc.), warm baths, massage, electricity (galvanism), and tonics, such as iron, quinine, arsenic, etc.

DIFFUSE AND FOCAL DISEASES OF THE BRAIN.

HYPEREMIA OF THE BRAIN.

Active hyperemia of the brain occurs in the many and varied conditions that increase the force and rapidity of the heart's action, especially the acute infectious diseases, alcoholism and other intoxications, prolonged mental strain, and emotional disturbances; and it is a part of sunstroke, meningitis, and encephalitis. Although in these circumstances more blood passes through the brain in a given time, it is not definitely known that more than a normal amount is present at any one time (on account of the relative inexpansibility of the brain and the absence of local vasomotor control); nor is it likely that the condition of itself gives rise to any noteworthy symptoms; the cerebral symptoms that do occur are much more likely due to the initiating infectious or toxic factor.

Passive hyperemia of the brain occurs in conditions of general venous engorgement, such as develop in failure of the heart induced by any cause (myocardial, endocardial, pericardial, or chronic pulmonary disease), and local conditions, such as venous or sinus thrombosis, tumors and abscess of the brain, and tumors, aneurysms, and other factors obstructing the circulation in the neck. The *symptoms* consist of headache, with a feeling of general tenseness and perhaps pulsation, and of pressure behind the eyes, vertigo, tinnitus aurium, mental irritability and incapacity, etc. The diagnosis should always be made with circumspection, since the symptoms mentioned are more likely to be due to some cause (toxic?) other than mere congestion; and, assuredly, in the chronic cases. The *treatment* of such cases as may be recognized consists of attention to the underlying causative factor; the use of general depletive measures, especially calomel, or blue mass and saline cathartics, hot foot baths, counterirritation to the back of the neck; an ice-cap to the head; and the administration of the bromides, ergot, etc.

ANEMIA OF THE BRAIN.

Anemia of the brain may result from: (1) Acute or chronic, primary or secondary, anemia. The symptomatology and treatment are mentioned on page 365 et seq. (2) Disorder of the vasomotor influences acting in the splanchnic area and the skin. When, for instance, the vasomotor centre becomes inhibited, as may happen under the influence of emotions, or when it becomes of lessened functional activity, as may occur in infectious and toxic processes, the circulation in the brain may become readily disturbed. In the first mentioned case the abdominal vessels dilate, and the general blood pressure in consequence falling, the heart is no longer able to maintain an efficient circulation in the

brain, cerebral anemia ensues, and the patient faints; in the second mentioned case, a satisfactory cerebral circulation may be maintained in the recumbent posture, but sudden exertion, emotional disturbances, or the assumption of the sitting or erect posture, may similarly induce cerebral anemia and fainting. A like result may also follow the sudden liberation of excessive intra-abdominal pressure—withdrawal of ascites, removal of an abdominal tumor, etc. The treatment consists of the recumbent posture (to favor the ready access of blood to the brain) and the use of diffusible stimulants. (3) Localized anemia of the brain may occur in arteriosclerosis and in what may be interpreted as a condition of angiospasm; foci of temporary stasis of the blood are doubtless often associated. The symptoms consist of momentary inability to perform special cerebral acts, depending upon the area involved, such as think, speak, see, move certain muscles, etc. (page 482). These may or may not be associated with temporary (momentary) loss of consciousness. The conditions have been likened to intermittent claudication (page 493). The treatment is that of arteriosclerosis (page 493).

ŒDEMA OF THE BRAIN.

Œdema of the brain may occur: (1) In conditions associated with passive congestion; (2) in chronic nephritis, in which the œdema may be widespread or localized, is doubtless toxic in nature, and is answerable for some of the uremic manifestations; (3) in traumatic disorders, such as concussion and contusion; (4) about abscesses and tumors; and (5) in congenital atrophic disorders, in which the œdema and increased cerebrospinal fluid fills up potential spaces. The *symptoms*, except in chronic nephritis, are due to associated conditions rather than to the œdema per se.

CEREBRAL HEMORRHAGE.

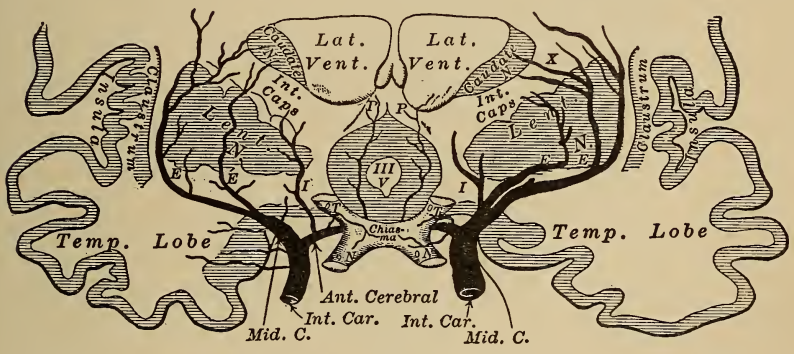
(*Apoplexy; A Stroke; Hemiplegia.*)

Etiology.—Hemorrhage into the substance of the brain is most common in male subjects after the fortieth year. The etiological factors are those of arteriosclerosis (page 489), especially syphilis, chronic alcoholism, chronic nephritis, excessive muscular work, plumbism, gout, diabetes, etc. The exciting cause of the hemorrhage is often some undue increase of blood pressure, such as may be provoked by exertion (running, lifting heavy bodies, straining at stool, etc.), or emotional disturbances; but in many cases the rupture of the bloodvessel occurs during quietude (as sleep)—in which event one may assume that the gradually weakened vessel finally becomes unable to sustain the ordinary blood pressure of that individual. Cerebral hemorrhage, however, may occur into tumors; it may develop in conditions of blood dyscrasia, such as per-

nicious anemia, leukemia, purpura, scurvy, septicopyemia, etc.; and occasionally it is observed in children.

Pathology.—In about three-fourths of the cases the hemorrhage occurs from one of the several branches of the middle cerebral (sylvian) artery, which, penetrating the anterior perforated space, supply the corpus striatum and the internal capsule, especially the lenticulostriate branch (Duret's artery; Charcot's artery of cerebral hemorrhage) and the lenticulothalamic branch (Fig. 22). In about one-fourth or more of the cases the hemorrhage occurs variously from any of the many branches of the cerebral arteries, and into the brain cortex, the peduncles, the pons, the medulla, or the cerebellum. The lesions consist of the ordinary changes of advanced arteriosclerosis (page 489); small, so-called miliary aneurysms are common, and rupture of these is the usual cause of the bleeding. Rarely, as in toxic states and the various blood dyscrasies, there may be an undue permeability of the vessel walls and an undue fluidity of the

FIG. 22



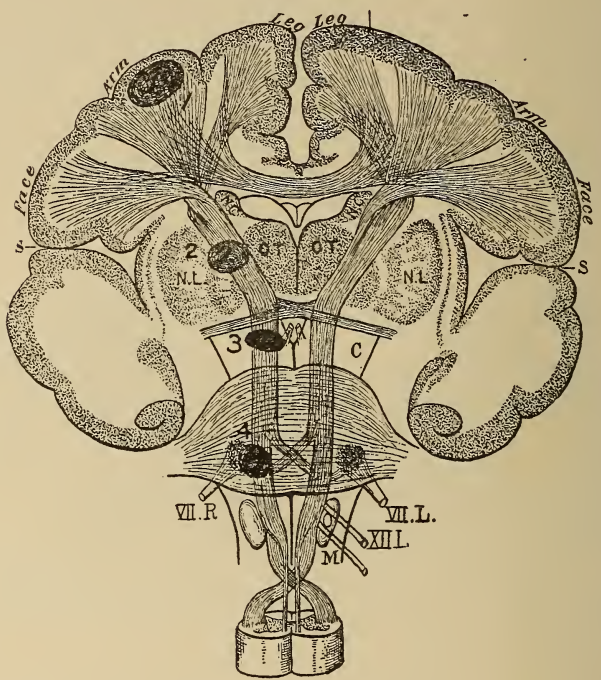
The distribution of the bloodvessels to the internal capsule: *E*, the lenticulostriate arteries (external arteries of the corpus striatum). *I*, the lenticulothalamic arteries (the internal arteries of the corpus striatum). (Duret.)

blood. The hemorrhage may be large or small; in most cases it is large enough to destroy the outer portion of the lenticular nucleus, the internal capsule, and the thalamus; but it may extend to the caudate nucleus and the insula, and in extreme cases it may involve almost, if not quite, all of the hemisphere and invade the lateral ventricles. In other cases the hemorrhage may be small—pinhead in size, for instance—but such a small hemorrhage, if occurring in the pons or medulla, may be attended by very serious consequences, even death. The blood, soon after being poured out, clots; ultimately the clot may be entirely absorbed, and represented by a scar, or a cyst may be the residual manifestation. Secondary degeneration of those fibers of the upper motor neuron deprived of continuity with their cell bodies in the cerebral cortex ensues.

Symptoms.—In a few cases symptoms described as premonitory are noted. Men of an apoplectic habitus (short, rather stout body, with a short, thick neck, prominent arteries, and ruddy complexion) are still

believed by some persons to be predisposed to cerebral hemorrhage; but the question is solely one of the cerebral arteries and the mode of life. In these, as well as other subjects with sclerotic vessels and high blood pressure, there may be attacks of headache, vertigo, and transitory incapacities (mentioned on page 862); or slight paresis or paralysis involving the cranial nerves (especially ptosis, strabismus, or disturbances of vision), and due perhaps to small hemorrhages, may ensue; or actual

Fig. 23



Scheme of the motor tract to show the effect of hemorrhagic or softening (thrombotic or embolic) lesions at different positions: 1, cortical or subcortical lesion, causing monoplegia of the left arm; 2, capsular lesion, causing left hemiplegia; 3, crus lesion, causing left hemiplegia and right third nerve palsy; 4, pons lesion, causing alternating paralysis of the right face and the left arm and leg; S, sylvian fissure; OT, optic thalamus; NL, lenticular nucleus; C, crus; NC, caudate nucleus; VII, facial nerve; M, medulla. (Starr.)

hemorrhages may be detected in the retina; or paresthetic phenomena (numbness and tingling), or choreiform or other movements may occur in an arm or a leg.

In the great majority of cases, however, the apoplectic attack comes on suddenly, without warning, The patient, in apparently good health, while sitting quietly, or walking, or after some exertion or emotion, suddenly becomes dizzy and falls to the ground unconscious; he may vomit and exhibit some convulsive movements. In the usual cases, those due to hemorrhage from the *lenticulostriate artery* or *into the internal capsule* (Fig. 23), and in any event if the hemorrhage is of at least moderate size,

the patient is soon found to have a livid or ashen-gray countenance, bathed in sweat, a full, bounding, rather infrequent pulse under increased pressure, and a normal or subnormal temperature; to be breathing slowly or stertorously, and "flapping" one cheek (paralysis), or exhibiting the Cheyne-Stokes rhythm; and to be paralyzed on one side of the body, which, however, may be difficult to determine on account of complete muscular relaxation. In addition, the head and the eyes may be turned toward the side of the lesion in the brain (paralytic conjugate deviation); the pupils are likely to be unequal or dilated (especially on the side of the lesion), and to be inactive to light if the patient is in deep coma; and control over the vesical and rectal sphincters may be lost. In some cases the paralysis and other phenomena are noted only when the patient awakes in the morning; or, not awakening, he is found to have died of apoplexy during the night. In other cases the onset is slower: there may be at first only headache and vertigo, and perhaps vomiting, with gradual loss of power in the arm and leg; but in the course of a few hours or a day unconsciousness supervenes and deepens into coma—so-called ingravescent apoplexy, due often to slow leakage of blood into the external capsule, and its gradual breaking through into the internal capsule and perhaps the lateral ventricle.

One of several eventualities may ensue: (1) The patient may die soon, or within several hours of the onset of the apoplexy. (2) The coma may continue, or it may lessen somewhat and become associated with delirium and fever—102° to 104° (so-called febrile reaction)—and then, recurring, the patient may die at the end of a week or thereabouts. In some of the cases, neither improving much nor retrograding, a sudden increase of the symptoms may occur; muscular rigidity and perhaps convulsions may ensue, and spasmodic conjugate deviation of the head and the eyes away from the side of the lesion may supervene. In some of these cases sudden inundation of the ventricles is answerable for the finally fatal symptoms. (3) With more or less return of consciousness, although not complete mental clearness, fever may set in; trophic changes may ensue, especially bed sores and sloughing on the paralyzed side; congestion of the bases of the lungs (in some way related to the cerebral disorder) may supervene; and with bronchopneumonia or septicopyemia the patient may die in two or three weeks. (4) The coma may gradually subside and the paralysis become obtrusive. This is unilateral, and may involve the leg, arm, and one-half of the face and tongue (complete hemiplegia), or only one of these parts (sometimes called partial hemiplegia). The paralysis at first is flaccid, and is associated with lessened or absent deep reflexes; soon an early rigidity may develop and be associated with increased reflexes and the Babinski sign. The speech is indistinct; aphasia (page 840) may occur in left-sided lesions. There may be slight or no sensory phenomena.

Hemorrhage into the crus gives rise to paralysis of the face, arm, and leg of the side opposite the lesion, and paralysis of the oculomotor nerve of the same side (syndrome of Weber; crossed or alternate hemiplegia).

There may also be contralateral hemianesthesia from extension to the tegmentum.

Hemorrhage into the pons and medulla is usually fatal within a few hours; but, if the patient survives, there is likely to be convulsions, deep coma, contracted pupils, and high fever; and in many cases bilateral paralysis, because of involvement of both motor tracts. If the hemorrhage is small, there is contralateral hemiplegia and homolateral disturbance with the functions of the nerve nuclei therein located (crossed hemiplegia); loss of sensation in the area of trigeminal distribution; paralysis of the muscles supplied by the facial nerve; or the phenomena described under acute bulbar paralysis (page 850).

Hemorrhage into the cerebellum gives rise to few symptoms significant in themselves. Coma, convulsions, and vomiting are common; paralysis may result from pressure on the nuclei or tracts in the medulla or pons; characteristic symptoms, such as vertigo, staggering gait, and inability to maintain muscular balance and the erect posture, can scarcely be ascertained. Death usually ensues.

Occasionally, after the lapse of several weeks all evidences of the apoplexy and paralysis have disappeared and the patient appears to be as well as before the attack. In the majority of cases, however, at the end of a month or six weeks the patient exhibits the permanent results of the hemorrhage—the secondary symptoms, or the chronic stage. As a rule, the paralysis is less than at first; some power has returned in the arm and the leg—in the leg first, and to a greater degree; the paralysis of the face and the tongue and any sensory disturbances originally present may have wholly disappeared. In the usual cases (hemorrhage into the internal capsule), however, more or less unilateral paralysis of the upper motor neuron type persists. This is most marked in the arm and the leg; muscles that act bilaterally, such as those of the face (wrinkling the forehead, frowning, closing the eyes) and of the trunk (respiration, etc.), are likely to show relatively slight impairment of function, since they doubtless may be innervated by stimuli from one side of the brain; and movements associated with emotional disturbances (langhing and crying) also are usually well executed, although the patient may not be able voluntarily to move the angle of his mouth. In addition, rigidity (so-called late rigidity) of the affected muscles develops and usually progresses to deformities (contraction of the flexors and adductors overcoming the extensors and abductors); in walking, the great toe is dragged along the ground, and the leg, as a whole, circumducted (swung on an arc of a circle from the hip); the deep reflexes are exaggerated, and ankle clonus and the Babinski sign become apparent; there is little, if any, wasting of the muscles aside from what may be attributed to disuse. Sometimes there is persisting hemianesthesia (involvement of the posterior part of the internal capsule), and rarely pain (irritation of the sensory neuron). Athetoid and choreiform movements may develop in the paralyzed limbs (posthemiplegic chorea and athetosis). In most cases there is at best slight impairment of mentality; in many cases

mental irritability, emotional disturbances on slight provocation, failure of memory, etc., supervene, and dementia may occur.

Diagnosis.—At the time of the apoplectic attack one must differentiate it from coma due to other causes, such as: (1) Embolism and thrombosis. Apoplexy is often employed as an inclusive term, comprising hemorrhage and softening (embolism and thrombosis); but thrombosis is suggested by gradual onset of the paralysis and coma, both of which may be transitory; by a history of syphilis or of another infection recently passed through; and by conditions of the blood generally that favor clotting. Embolism is suggested by a young subject and vegetative endocarditis or other disorder whence an embolus may be derived. (2) Uremia. Renal disease (cardiovascular-renal disease) is often present in those the subject of apoplexy, so that it may be difficult to make a differential diagnosis; but uremia is suggested by albumin and casts in the urine, convulsions, very high blood pressure, albuminuric neuroretinitis, the absence of hemiplegia, and a history of previous attacks without consecutive paralysis. But now and then fatal cases of hemiplegia are encountered in which only nephritis and œdema of the brain and meninges are found at the necropsy. (3) Diabetic coma—which may be distinguished by ketonuria, perhaps glycosuria, fruity odor to the breath, the characteristic Kussmaul dyspnœa, and the absence of hemiplegia. (4) Alcoholic coma (obvious alcoholism, alcoholic habits, less deep coma, and absence of hemiplegia or other localized paralysis). (5) Opium poisoning (contracted pupils, infrequent respirations, no convulsions, no paralysis).

Prognosis.—Death may occur on the first attack, but recovery, with more or less permanent incapacity, frequently ensues. Succeeding attacks are likely to occur, and with each the tendency to fatality increases.

Treatment.—It is important, first, to determine whether the "apoplexy" is due to hemorrhage or to thrombosis or embolism. In hemorrhage the patient should be kept quiet, with the head high and the neck free. If breathing is obstructed, he should be turned on his side, to obviate obstruction of the pharynx by the paralyzed tongue. To reduce the blood pressure and promote stilling the bleeding, ice may be applied to the head and hot bottles to the feet, or a hot mustard foot bath may be given; and venesection should be performed; or one may give aconite, veratrum viride, or nitroglycerin, and open the bowels freely (calomel, croton oil, eletarium). But Crile and Cushing have shown that a high arterial pressure may be conservative, inasmuch as this tends to supply a sufficiency of blood to the vital centres in the medulla, which otherwise—that is, under the influence of a rising intracranial pressure—would rapidly become anemic and exhausted. Therefore, in cases with marked and rising blood pressure (250 mm. Hg.—indicative of increasing intracranial pressure), especially if associated with an infrequent pulse, Cushing recommends, and has practised with good results, trephining the skull and evacuating the blood clot. This is particularly indicated, of course, in meningeal hemorrhage, but it has been suc-

cessfully done in cerebral hemorrhage. In the ingravescent type of apoplexy compression, even ligation of the carotids, has been practised.

Otherwise, the treatment in the early days consists of good nursing, rest in bed for ten days or two weeks, the prevention of bed sores, attention to the position of the paralyzed limbs which may be enveloped in cotton wool, catheterization if necessary, and after the first day or two a light, nutritious diet. Stimulants and heart tonics should be avoided, unless cardiovascular collapse develops. In addition, after the lapse of ten days or two weeks, the paralyzed limbs should be rubbed for ten to fifteen minutes twice daily, to maintain nutrition and lessen contractures, and at the end of three or four weeks the faradic current may be applied—especially to the extensors and abductors—which are likely to be soon overcome by the contracting flexors and adductors. Iodide of potassium is often given, in the hope that it tends to promote absorption of the clot. It is of value in syphilitic cases, and in the event of recent syphilis may be combined with mercury. In the course of time resort to surgical intervention, tendon cutting or transplantation, and nerve transplantation, may be advisable to overcome disabling contractures.

CEREBRAL THROMBOSIS.

(*Cerebral Softening.*)

Etiology.—Thrombosis of the cerebral vessels occurs under conditions that favor clotting elsewhere—the chief of which are arteriosclerosis and a weak or slow circulation. It most commonly follows syphilitic endarteritis, and occurs in middle and advanced life. It may develop within small or fair-sized aneurysmal dilatations of the cerebral vessels, and after partial or complete obstruction by a clot. It may occur during the course of, or following, infectious diseases, and in conditions of blood impoverishment favoring clotting.

Pathology.—Thrombosis is most common in the middle cerebral artery (supplying the motor cortex as well as the corpus striatum) and in the basilar arteries. The lesions are those of thrombosis in general, followed by an area of infarction, which may be red (or hemorrhagic), but often is pale (or anemic). The softened and necrotic area becomes replaced by a scar, or a cyst ensues, and the fibers deprived of continuity with their nuclei, degenerate.

Symptoms.—The symptoms vary with the mode of onset and the region of the brain involved. Even extensive softening may be found without noteworthy symptoms (involvement of so-called silent areas of the brain). In some cases the onset is sudden and quite like that of hemorrhage. In many cases, however, the onset is gradual, and may be preceded by certain prodromes attributable to temporary anemia of certain regions of the brain, such as temporary inability to think, speak, see, remember, or to move certain muscles (ptosis, strabismus, etc.); or

numbness or tingling in an arm or a leg. Out of these there may develop gradually progressing paralysis of an arm, a leg, half of the face or the tongue, or a partial or complete hemiplegia (depending upon the part of the brain involved—small or large areas of the cerebral cortex or the internal capsule, etc.); and these may or may not be attended by loss of consciousness, which, if it does ensue, is gradual in development. Both the unconsciousness and the paralysis are likely to lessen considerably, since, although usually looked upon as end arteries, some considerable collateral circulation may become established; but a permanent, partial or complete hemiplegia is the rule, as in cerebral hemorrhage. Aphasia (page 840) may ensue in left-sided lesions. Thrombosis of the basilar artery and its branches gives rise to lesions similar to those of hemorrhage into the pons (page 866). Thrombosis of the posterior cerebral artery gives rise to hemianopsia and sensory aphasia.

Diagnosis.—The diagnostic data are mentioned in connection with those of hemorrhage (page 867).

Prognosis.—The outlook in general is rather better than in hemorrhage, but the tendency to thrombosis is likely to remain, and mental deterioration, loss of memory, etc., are likely to ensue.

Treatment.—The treatment comprises efforts to ward off an attack, the imminence of which is suggested by the prodromes mentioned; these consist of measures useful in arteriosclerosis (page 493) and syphilis (page 190), and cardiac stimulants, such as alcohol, ammonia, ether, caffeine, strophanthus, nitroglycerin, and digitalis (in small doses, and cautiously). The treatment of the developed attack should consist of rest, with the head low, and the aforementioned cardiac stimulants. Care should be taken not to depress the circulation and thus favor additional clotting. The general hygienic management and the use of massage and electricity are as has been mentioned in connection with cerebral hemorrhage (page 867).

CEREBRAL EMBOLISM.

(*Cerebral Softening.*)

Etiology.—Cerebral embolism occurs most commonly in cases of chronic recurring endocarditis of the mitral and aortic valves, and is therefore most common in young subjects, especially women. Occasionally it occurs in other conditions in which emboli gain access to the general circulation (clots from an aneurysm, atheromatous plaques, etc.); and in conditions of arterial pyemia, but since these emboli are infected, a brain abscess ensues.

Pathology.—Emboli reach the brain more commonly by the left than by the right carotid artery, and the lesions therefore are usually left-sided. They consist of plugging of a vessel, usually a branch of the middle cerebral artery, and an area of softening analogous to that which follows thrombosis. More or less widespread thrombosis may follow the embolic plugging of the vessel.

Symptoms.—The symptoms are sudden in onset, although they vary with the site of lodging of the embolus. Usually the common symptoms of cerebral hemorrhage develop, and the patient is soon found to be hemiplegic and aphasic. Convulsions may occur in case the cortex be involved.

Diagnosis.—The diagnosis is to be based upon the occurrence of hemiplegia or other paralysis in a young subject with endocarditis or other disorder whence an embolus may be derived. The differential diagnosis is like that of hemorrhage (page 867).

Prognosis.—The prognosis is rather better than in hemorrhage, but the likelihood of recurrence must be borne in mind if the initiating factor persists.

Treatment.—The treatment is that of thrombosis (page 869) plus the treatment of the primary cardiac or other disorder.

THROMBOSIS OF THE CEREBRAL SINUSES.

Etiology.—Thrombosis of the cerebral sinuses may be primary or secondary. The primary cases, which are usually simple or non-infected, may result from anemic states (chlorosis and severe anemias in general); cachectic states, such as diarrhœal and other wasting diseases of children, and advanced carcinoma, Bright's disease, etc., in adults; and infections such as typhoid fever, tuberculosis, etc. The secondary cases, which are usually infected, result from extension of disease of contiguous structures, especially middle-ear disease, brain abscess, fracture of the skull and subsequent infection, and extension of suppurative processes outside the skull (erysipelas and other pyococcic infections).

Symptoms.—*Thrombosis of the lateral sinus* (usually associated with middle-ear disease) gives rise to pain, tenderness, and œdema over the mastoid, fulness of the regional veins, often induration of the internal jugular vein from extension of the thrombosis, and the general evidences of septicopyemia (chills, fever, sweats, and polynuclear leukocytosis), and often delirium.

Thrombosis of the longitudinal sinus (which occurs usually in anemic and cachectic states, after typhoid fever, etc.) may not cause any obtrusive symptoms, the symptoms of the primary disease being predominant; but headache, mental dulness, convulsions, and various paralyses, depending upon the portion of the motor cortex involved, may ensue. In the absence of infection there are no general symptoms due to the thrombosis itself.

Thrombosis of the cavernous sinus (which usually follows infections of the nasal or orbital cavity) may give rise to frontal or lateral headache, œdema of the orbital tissues, exophthalmos, conjunctival ecchymosis, ulceration of the cornea, papillœdema, paralysis of the ocular muscles, etc.

Treatment.—The treatment of the infective cases is surgical, and should not be delayed. The treatment of the anemic and cachectic cases is that of the primary disorder, and the use of the iodides, iron, quinine, strychnine, etc.

ACUTE HEMORRHAGIC ENCEPHALITIS.

(*Acute Hemorrhagic Cortical Encephalitis; Inferior and Superior Polioencephalitis; Cerebellar Encephalitis.*)

Etiology.—Local or diffuse inflammation of the brain substance occurs under varying circumstances: (1) It may follow fractures of the skull. (2) It occurs, to a greater or less extent, in all cases of meningitis. (3) It may develop about foci of hemorrhage and softening (embolism and thrombosis). (4) It may follow acute infections, such as diphtheria, scarlatina, influenza, pneumonia, etc. (5) It has been attributed to alcoholism, ptomaine poisoning, lead poisoning, carbon monoxide poisoning, etc. (6) It may occur as an independent disorder, perhaps an acute infectious disease, involving the cortex or the nuclei of cranial nerves. In this, the superior and inferior polioencephalitis of Wernicke, the lesions consist of acute hemorrhagic inflammation of the gray matter about the aqueduct of Sylvius; these have been described under ophthalmoplegia (page 851). In other cases the lesions may be confined to the motor cortex, the *acute infantile cerebral palsy* of Strumpell; a similar condition may be found in adults. In other cases the inflammatory process may be limited to the area of distribution of a single arterial branch, or it may involve corresponding areas of both hemispheres; or it may be disseminated in the cerebrum and the cerebellum. The lesions consist of foci of congestion, hemorrhage, and softening, involving especially the gray matter; in many respects the condition is the cerebral homologue of acute poliomyelitis.

Symptoms.—The symptoms are often equivocal; often for a time they are those of a general infection only, with headache, delirium, coma, fever, etc., but to these, focal symptoms are commonly added, varying with the seat of the lesions. There may be paralysis of an arm or a leg or of any of the muscles supplied by the cranial nerves, aphasia, anesthesia, convulsions (cortical or Jacksonian in type), etc. Death may ensue within several weeks, or, the acute symptoms subsiding, the condition may become chronic and more or less mental deterioration (imbecility) may persist.

ABSCESS OF THE BRAIN.

(*Acute Suppurative Encephalitis.*)

Etiology.—Abscess of the brain is most common in young male adolescents and adults. The causes are: (1) Traumatism of the head,

usually fracture of the skull, and subsequent infection. (2) Extension of suppuration from adjacent structures, especially middle-ear disease, with involvement of the mastoid, nasal suppuration, and necrosis of the bones of the skull. (3) Transport of infection from distant regions, such as lung abscess, bronchiectasis, empyema, (4) General septicopyemia. The infectious agents comprise staphylococci, streptococci, pneumococci, Bacillus pyocyaneus, Bacillus influenzæ, Bacillus tuberculosis, Nocardia, etc.

Pathology.—There may be one or many abscesses. Usually there is only one, which varies in size from 2 to 20 or 30 cm. in diameter, and is most common in the temporosphenoidal lobe or the cerebellum (on account of the proximity of the bones of the ear); but it may be situated in the frontal, parietal, or occiptal lobe. The pus is often greenish and has a foul odor. In acute cases there is likely to be considerable congestion and œdema of the surrounding brain tissue; in chronic cases there may be a more or less well-defined area of limiting neuroglial tissue, or the surrounding brain tissue may be rather widely infiltrated with pus. The multiple abscesses are usually small, often, indeed, microscopic in size; larger ones may form from coalescence of smaller ones.

Symptoms.—Corresponding with the mode of development of the abscess, the symptoms may come on acutely or insidiously. The *acute* cases usually follow a trauma or rapidly progressing middle-ear disease. The chief symptoms consist of general nervous phenomena, such as severe headache, vertigo, vomiting, delirium, mental dulness, stupor or coma, and often papillœdema and slow pulse; focal nervous symptoms depending upon the situation of the abscess (see cerebrospinal localization, page 831); and the systemic manifestations of septicopyemic states, such as chills, fever, sweats, and polynuclear leukocytosis. In the *chronic* cases the abscess may slowly attain considerable dimensions without occasioning symptoms—so-called latent stage, which is the more likely to be present if the abscess develops in one of the so-called silent areas. Symptoms, when they ensue, are like those just mentioned, but less severe; regression and exacerbation of the symptoms may occur from time to time, and the exacerbations may be associated with convulsions. In cases of general septicopyemic or of local suppurative processes elsewhere, the general and local symptoms due to these may overshadow those due to the brain abscess—which may not be recognized.

Diagnosis.—The diagnosis depends upon an etiological factor, the general and local evidence of disease of the brain, and the systemic signs of pyogenic infection. Brain tumor (page 874) and meningitis (page 830) must be excluded.

Prognosis.—Unless evacuated, the abscess causes death by destroying or compressing vital regions of the brain, or by provoking suppurative meningitis or general septicopyemia.

Treatment.—The treatment is wholly surgical, and consists in evacuation and thorough drainage of the abscess. This should be promptly resorted to, and is often followed by recovery.

TUMORS OF THE BRAIN.

Etiology.—Tumors of the brain are more common in males than in females, and they occur at all ages, but, as a rule, before rather than after the fortieth year; they are comparatively common in young subjects and children. Some cases seem to follow traumatism to the head; but otherwise the etiological factors are those of tumors in general, and are as ill understood.

Pathology.—Tumors of the brain may be primary or secondary, and benign or malignant, but the so-called benign tumors (the type of tumor benign in other parts of the body) becomes in the brain more or less malignant and leads to death, because of the implication of vital structures. The common tumors are sarcoma, glioma (gliosarcoma), endothelioma, carcinoma, and fibroma; but to these one must add the tubercle (solitary tubercle, tuberculoma), gumma, and cyst, since these formations, especially tubercle and gumma, are very common in the brain, and there are essentially tumors in their growth and manifestations. The tubercle (the most common) is usually secondary to tuberculosis elsewhere; it is generally single, but may be multiple; it varies in size up to that of a walnut, or larger; it occurs especially in young subjects; and it develops, as a rule, from the meninges (associated meningitis), although it may be found within the brain substance—near the cortex, at the base, or in the cerebellum. The gumma is usually multiple; it develops, as a rule, from the meninges; it is found most commonly at the base of the brain or in the cortex, and comparatively rarely within the substance of the cerebrum or cerebellum. The endothelioma and fibroma develop from the meninges or nerve sheaths and compress the brain. The glioma (gliosarcoma) develops from the neuroglia and infiltrates the brain substance. Other sarcomas, as well as carcinomas, may develop in any part of the brain as secondary metastatic nodules. Cysts may be parasitic (rare) or simple (softening cysts), and occur anywhere in the brain. Rarer tumors comprise psammoma, cholesteatoma, lipoma, osteoma (from the skull), angioma, aneurysm, etc.

Symptoms.—The symptoms vary with the variety, situation, and rapidity of growth of the tumor. Rarely, a tumor is found after death, in the absence of noteworthy symptoms during life; usually it has occupied one of the so-called "silent areas." In the great majority of cases, however, there are characteristic symptoms; of these, one group, the general symptoms, develop irrespective of the situation of the growth; another group, the focal symptoms, depend upon the situation of the tumor. The important general symptoms comprise headache, vertigo, vomiting, and papillœdema and optic atrophy. Headache is almost always present, and once developed rarely disappears, although it may vary in intensity from time to time. It may be dull and aching; or a sensation of fulness and compression; or it may be extremely severe and paroxysmal, especially at night (syphilis). Sometimes it is localized

—which rarely is of diagnostic significance, although, if associated with local tenderness, it may suggest the situation of a gumma and local meningitis. The headache is variously due to increased intracranial pressure, stretching of the meninges, pressure upon and irritation of sensory nerves (especially the fifth), localized meningitis, etc. Vertigo is often an intractable symptom, sometimes occurring spontaneously, and often set up by changes in posture. It may occur in association with tumors of any part of the brain, but is most common in tumors of the base, or the cerebellum. Vomiting is common; it has no relation to taking food; it is usually unattended with nausea, the contents of the stomach, as a rule, being suddenly violently ejected without antecedent warning; it may be induced by sudden movements of the head (which may also cause vertigo). It is most common in tumors of the base of the brain—beneath the tentorium (great increase in intracranial pressure). Papillœdema (choked disk) in many respects is the most important, and, with the other symptoms, the most trustworthy manifestation of brain tumor. It occurs in at least 90 per cent. of the cases, and is most common in tumors of the base of the brain and cerebellum; the condition is usually bilateral; in some cases it is more marked on the side of the lesion; in other cases on the other side. The rapidity of its progress is sometimes an evidence of the rapidity of growth of the tumor (perhaps, more correctly, of the rapidity of increase of intracranial pressure); often there is associated neuroretinitis. Optic atrophy is the usual sequel.

There are other more or less important general symptoms, such as general irritability, apathy, somnolence (sometimes insomnia), melancholia, even dementia or mania. General convulsions sometimes occur, and are of importance, since they may be mistaken for epilepsy, and thus lead to overlooking of the brain tumor, at least for some time. If well studied, they may reveal a definite, well-localized onset (Jacksonian or focal epilepsy), of value in localizing the tumor. Syncopal and apoplectiform attacks sometimes occur; the latter may be due to hemorrhage into the tumor. The pulse is usually slowed, and may be irregular; toward the end of life it may become rapid. The temperature may be normal or subnormal; occasionally there is fever (inflammatory lesions in the brain or meninges). Polyuria and glycosuria are sometimes observed (increased pressure or irritation of the medulla); and the patient may emaciate.

The focal or localizing signs of tumors of the brain are those mentioned under cerebrospinal localization (page 831).

Diagnosis.—The diagnosis may be based upon any two of the important general symptoms (headache, vertigo, vomiting, and papillœdema) and such localizing signs as may be detected by a careful examination. Since papillœdema may be present for some time and in notable degree, with little or no impairment of eyesight, the eyegrounds should be examined in all cases in which the possibility of brain tumor exists. Brain abscess may be excluded by the absence of the etiological factors of abscess (local trauma, middle-ear disease, etc.), the less rapid progress

of the symptoms, and the absence of general evidences of infection (chills, fever, sweats, and polynuclear leukocytosis). Meningitis (page 828), cerebral hemorrhage (page 862), cerebral softening (page 868), and sometimes uremia (page 759) must also be excluded.

Prognosis.—The prognosis depends upon the nature and situation of the growth and its amenability to surgical removal. Gummas may be absorbed by antisyphilitic treatment. In other tumors the average duration of life is about two years or less. Death may occur suddenly, in an apoplectiform attack for instance, or during general convulsions, etc.

Treatment.—The possibility always exists that the tumor may be syphilitic; and since less than 10 per cent. of the cases are at any time amenable to surgical removal, antisyphilitic treatment should be tried, especially in all cases of doubt. Preference should be given to inunctions of mercury and the concurrent and subsequent use of the iodides in very large doses (150 to 250 grains, 5 to 8 grams, daily). It is useless to continue the treatment with mercury if no improvement occurs within four to six weeks. Marked improvement, even cure, has been reported from the long-continued use of the iodides, even in supposedly non-syphilitic cases; at all events, the headache is thus often notably relieved. The vertigo and vomiting may be mitigated by the bromides. Convulsions call for morphine and atropine hypodermicly.

Otherwise the treatment is surgical and has two objects in view—the removal of the growth and the relief of symptoms. The decision for surgical intervention should not be too long delayed, and the operation should be undertaken if the focal signs indicate accessibility of the tumor. In the event of inaccessibility, the operation known as decompression may be undertaken. It is designed to relieve intracranial pressure, by removing a portion of the skull, and it frequently results in lessening of the headache, vertigo, vomiting, and papillœdema. Some temporary relief of the increased intracranial pressure may also be effected by lumbar puncture.

HYDROCEPHALUS.

Hydrocephalus is a condition characterized by the accumulation of an increased, and often very large, amount of serous fluid within the ventricles of the brain. This condition is sometimes spoken of as internal hydrocephalus, to distinguish it from external hydrocephalus, a collection of fluid between the brain and the dura mater. This occurs in atrophy of the brain (hydrocephalus ex vacuo), in advanced life, and after hemorrhages, softenings, etc.

Internal hydrocephalus may be congenital or acquired. The congenital cases may be observed at birth, when the enlarged head may interfere with parturition, or it may not become apparent until the child is some months or a year old. The condition may occur in several members of the same family. The real cause is not known, although it is

likely some vice of development or a fœtal meningitis obstructing the foramen of Majendie and leading to the intraventricular accumulation of fluid. The acquired cases may result from an ependymitis with marked serous exudation, a condition which Quincke believes to be angioneurotic in nature (Quincke's angioneurotic hydrocephalus); or they may be due to inflammatory (meningitic) or pressure (tumor) obstruction of the passage from the third to the fourth ventricle or of the foramen of Majendie.

In the congenital cases and those developing in early life the head may be slightly or enormously distended. The bones of the skull are separated and the fontanelles widened; the forehead overhangs the face, the eyes are recessed, and the bones of the face seem small (only relatively small in comparison with those of the skull). The brain is enlarged, the ventricles more or less distended, and the cortex, as a rule, correspondingly atrophied. Although rarely considerable intellectual development occurs, the mentality, as a rule, is variously impaired up to complete idiocy. Death usually occurs in childhood, but the patient may reach adult life. The cases developing in adult life can scarcely be recognized with certainty; the hydrocephalus is usually only an incident in the course of the disease to which it is due.

DIFFUSE AND FOCAL DISEASES OF THE SPINAL CORD.

VASCULAR DISORDERS OF THE SPINAL CORD.

Hyperemia, anemia, and œdema of the spinal cord, speaking generally, occur under circumstances like those that occasion similar conditions in the brain (page 861 et seq.). They often exist without noteworthy clinical manifestations. The symptoms attributable to posterolateral or toxic combined sclerosis, which is rather common in grave anemias, are mentioned on pages 375 and 858. Embolism and thrombosis, and consequent softening of large areas (myelomalacia), are comparatively rare in the spinal cord; but thrombosis of fine bloodvessels is quite common in the arteriosclerosis of advancing life, and is found also in conditions of choreiform and athetoid movements, paralysis agitans, etc. Hemorrhage into the spinal membranes (hematorrachis) is mentioned on page 826.

HEMORRHAGE INTO THE SPINAL CORD.

(*Hematomyelia; Intraspinal Hemorrhage.*)

Etiology.—Hemorrhage into the spinal cord may be due to traumatism (fracture-dislocation, acute flexion of the cervical vertebræ without fracture or dislocation), to severe convulsive diseases, such as tetanus, or it may occur into an area of the cord the seat of antecedent disease, such as tumor, myelitis, syringomyelia, etc.

Pathology.—The hemorrhage may be very small (microscopic) and multiple, or single and very large, infiltrating a considerable length of the cord. Usually it occurs into the gray matter, which in consequence is most destroyed; but more or less laceration and secondary disorganization of the white matter may also be found.

Symptoms.—The symptoms depend upon the seat and extent of the hemorrhage. Usually there is sudden onset of paralysis, attended by more or less pain. The hemorrhage occurring high up (in the cervical region) may occasion paralysis of all four extremities, and may soon lead to death (involvement of the medulla). In other cases only the arms or the legs may be involved; in some cases there is a more or less outspoken and typical Brown-Séquard paralysis (page 838). If recovery ensues, there is more or less wasting of the muscles innervated by the cells in the anterior cornua (the usual seat of the hemorrhage) of the segment or segments involved; this is commonly more marked on one side of the body than on the other, and is associated with varying sensory disturbances—usually tactile hyperesthesia on the side of most marked paralysis, and impaired pain and temperature sensibility, with preserved tactile sensibility, on the opposite side. In most cases there is also more or less spastic paralysis of one or both legs (more marked in one than in the other), since the hemorrhage usually involves the pyramidal tracts (upper motor neuron) primarily or secondarily.

Diagnosis.—The diagnosis is comparatively easy in traumatic cases, and, in any event, if the symptoms are sudden in onset (which always suggests hemorrhage). Impaired pain- and temperature sensibility, with unimpaired tactile sensibility, suggests involvement of the gray matter (which is usually due to hemorrhage). The presence of antecedent disorders (tumor, myelitis, syringomyelia), to which the hemorrhage may be secondary, must be determined by eliciting the signs significant of these conditions.

Prognosis.—The outlook depends upon the seat and extent of the hemorrhage, and the presence or absence of associated lesions. In uncomplicated cases recovery usually ensues, but more or less permanent paralysis and disability persists.

Treatment.—The treatment consists of enforced rest; perhaps in the early days the application of an ice-bag to the spine (of questionable utility); and attention to the general hygienic conditions (feeding, prevention of bed sores, catheterization, enemas, etc.). Pain may require the use of morphine; general nervousness, the bromides, chloral, etc. Painful contractures of the muscles may be relieved by massage and warm applications. The iodides have been recommended to promote absorption of the clot.

In traumatic cases radiographic examination may be resorted to to determine the nature of the lesion, and fixation apparatus may be applied if necessary to maintain a desirable position. The question of operation must be considered. It is always wise to postpone this until the shock has passed; and in some cases, even for a week or thereabouts,

to distinguish between symptoms due to compression or concussion and those due to permanent damage to the cord. In the event of total transverse destruction of the cord an operation is useless. But operation should not be too long delayed; beneficial results are much more likely to follow early rather than late operations.

CAISSON DISEASE.

(Driver's Paralysis; Compressed Air Disease; The "Bends.")

Caisson disease is a form of paralysis characterized especially by paraplegia that develops in persons who have worked under increased atmospheric pressure, upon their return to the normal pressure of the earth's surface.

Etiology.—While the disorder may develop in perfectly healthy men, it is much more common in those in poor health, from any cause; in alcoholic, cardiac, nephritic, and obese subjects; and in "green hands" rather than in those who have worked for some time under increased pressures.

Pathology.—The conspicuous lesions consist of air emboli in the veins and capillaries of the spinal cord, and foci of hemorrhage and softening. The disease is commonly attributed to concentration and compression, in the tissues of the body, of the normal gases of the atmosphere, caused by the abnormally great pressures within the caissons, etc., and to the sudden release of these compressed gases upon the subject passing rapidly from a high to a low atmospheric pressure. The condition, therefore, is due to the sudden expansion of gases in soft tissues, the brain and the cord, contained within an unyielding bony framework. McWhorter, in fatal cases, found the right side of the heart filled with gas, of which 80 per cent. was nitrogen and 20 per cent. carbon dioxide; and he found no abnormal gases.

Symptoms.—The symptoms consist of the sudden development, immediately or several hours after the subject leaves the caisson, of severe pain in the back and legs, so that he is unable to move, doubles up (the "bends"), and falls to the ground. The pain is in the muscles—which are also tender to palpation and on passive motion. Pain in the joints (knees, ankles, hips, elbows, etc.) also may be complained of, and add considerably to the patient's disability. In addition, headache, vertigo, tinnitus aurium, deafness, vomiting, and marked prostration often ensue; or apoplectiform attacks, with hemiplegia or monoplegia, and perhaps aphasia, may develop; or a true paraplegia may supervene. Death may occur in coma.

Diagnosis.—The disorder is readily recognized from the relationship of the symptoms and the etiological factors.

Prognosis.—The prognosis is good in most cases. Recovery usually ensues within several days; but death, or more or less chronic invalidism may follow.

Treatment.—The disease can be considerably lessened by careful physical examination of subjects presenting themselves for work in caissons, and rejecting only those in perfect physical health; and by gradual decompression—that is, the use of air locks or pneumatic cabinets, in which the pressure is gradually reduced, so that the workers are gradually brought from the greatly increased to the normal atmospheric pressure. The developed disease is best treated by recompression—that is, subjecting the patient to increased atmospheric pressure and later gradually reducing it. Ergot in large doses has also been recommended.

MYELITIS.

Myelitis, or inflammation of the spinal cord, may be a diffuse or a circumscribed process; it may involve only the gray matter—a disorder discussed under poliomyelitis (page 270), and polioencephalitis (pages 850 and 851); it may involve a narrow vertical area of both white and gray matter—transverse myelitis, or it may occur in scattered areas throughout the cord—disseminated myelitis.

Etiology.—Acute myelitis may be a primary or secondary process, but in all cases it must be ascribed to the activity of infectious microörganisms (or their toxins), although an analogous condition of softening (myomalacia) may result from interference with the blood supply (thrombosis or embolism, or compression). In some cases myelitis seems to be in itself an acute infectious disease, but no specific organism has yet been discovered. In other cases it follows the more common infections, such as typhoid fever, smallpox, pneumonia, dysentery, pyococcic and gonococcic infections, syphilis, tonsillitis, etc., the organisms gaining access to the cord variously by way of the bloodvessels or the lymphatics. In other cases myelitis follows meningitis and neuritis, and tuberculosis or syphilis of the vertebræ and spinal meninges. It is said also to follow exposure to cold and damp, traumatism to the vertebræ, etc. It is most common in male subjects before the fortieth year (susceptibility to the common exciting causes).

Pathology.—The lesions at first consist of swelling, congestion, œdema, and softening of the cord, so that the gray and the white matter may be indistinguishable (especially in transverse lesions). Microscopicly there is vascular congestion, cellular and fluid exudation, minute hemorrhages, swelling and degeneration of the axons and their myelin sheaths, and vascular and other degenerations of the ganglion cells of the gray matter. Soon the softened area may take on a yellowish color, due to fatty degeneration of the elements within the inflamed zone. Ultimately, if the patient survives, with absorption and disappearance of the degenerated tissues, the neuroglia undergoes hyperplastic, and later cicatricial, changes, so that the zone of inflammation and softening becomes replaced by scar tissue. In transverse lesions secondary degenerations ensue, beginning within twenty-four hours: so-called

descending degeneration in practically all the columns of the cord, but especially conspicuous in the crossed and direct pyramidal tract, and elsewhere in the anterior and lateral columns (association fibers); and ascending degeneration in the posterior columns of Goll and Burdach, the direct cerebellar tract, and the anterolateral ascending tract of Gowers.

Symptoms.—The symptoms vary with the seat and character of the lesion. The onset may be abrupt (myomalacia from vascular occlusion or myelitis) or more insidious (usually myelitis). Some fever is usually present in the inflammatory cases. The symptoms in transverse lesions consist of those due directly to destruction of a part of the cord and of secondary manifestations due to blocking of impulses in the cord. The symptoms due directly to the lesion (destruction of the lower motor neuron) consist of loss of voluntary muscular control, flaccidity, and wasting, and the reactions of degeneration in the muscles innervated by the spinal segment or segments involved; loss of the deep reflexes represented in the destroyed portion of the cord (destruction of the reflex arc); and anesthesia and paresthesia in the area of peripheral distribution of the dorsal roots involved (destruction of the sensory neuron). The secondary symptoms, those due to blocking of motor and sensory impulses in the cord, consist of spastic paralysis of the muscles below the lesion (legs), with rigidity, and ultimate contractures and deformities, but no reactions of degeneration and no atrophy, aside from that which follows disuse (disorder of the upper motor neuron); increased knee-jerks, ankle clonus, and the Babinski sign; hyperesthesia or anesthesia (protopathic, epicritic, and deep sensibility) below the lesion; loss of control of the vesical and rectal sphincters; and vasomotor and trophic disturbances, especially coldness and cyanosis of the extremities, bed sores at points of pressure (sacrum, heels, etc.), thin, glossy skin, etc. Just at and above the segmental level of the lesion there is usually a zone of hyperesthesia (irritation of the dorsal roots), which, together with the upper limit of the area of anesthesia, form the best guide to the determination of the level of the lesion (see Plate I, page 803).

The symptoms of myelitis occur in varying combinations, due to variations in the lesions. The following may be distinguished: (1) *Transverse thoracic myelitis*, of which the symptoms are those just narrated. (2) *Transverse lumbar myelitis*, which differs only in the fact that the cells of the lower motor neurons innervating the legs are likely to be involved, so that instead of spasticity, flaccid paralysis, and atrophy of the muscles, with lost knee-jerks and the reactions of degeneration, ensue. (3) *Transverse cervical myelitis*, in which, in addition to the general symptoms mentioned, there is likely to be paralysis and wasting of the muscles of the hand (cervical enlargement), inequality of the pupils (disorder of the cervical sympathetic), hyperpyrexia, and death from respiratory failure (involvement of the cervical roots from which the phrenic nerve is derived). (4) Involvement of the *conus medullaris* is rare, but involvement may be recognized by paralysis of the vesical and

rectal sphincters and of the levator ani muscles, and anesthesia and other sensory disturbances of the buttocks, perineum, and external genitalia. (5) *Acute ascending myelitis or paralysis* (*Landry's paralysis*). This is undoubtedly an acute infection of the spinal cord, sometimes of the spinal cord and peripheral nerves, and perhaps also the bulb. It is characterized by fever and associated constitutional disturbances, and by a rapidly ascending paralysis, which, beginning in the legs, soon involves the trunk, the arms, and the muscles innervated by the bulb (especially the muscles of respiration), so that death usually results within a few days from respiratory or heart failure, or paralysis of the muscles of deglutition. Occasionally a recovery is reported. There may be few, if any, sensory changes; the bladder and the rectum may not be notably involved; and consciousness is usually preserved to the end. The nature of the disease is not well understood; all cases apparently included in this category are not alike. In some cases the reverse order of the onset and progress of the paralysis is observed—a descending paralysis. (6) *Disseminated myelitis.* In this varying combinations of motor paralysis and sensory disturbances may occur simultaneously or in succession in the legs, trunk, and arms; only a few muscles of a limb may show atrophy and the reactions of degeneration; and the disturbances of sensibility may be complete or incomplete in different parts of the limb.

Acute myelitis may lead to death within a few days or a few weeks, or, if the lesions are slight, the initial phenomena, the paralyses, may subside more or less and the patient pass into what is often called *chronic myelitis* (perhaps in some cases the symptoms develop so insidiously as to merit the term chronic from the beginning). There is spastic paraplegia, muscular rigidity, and consequent deformity of the legs, lessened or abolished sensibility, and loss of control over the vesical and rectal sphincters.

Diagnosis.—The diagnosis should be readily made by attention to the aforementioned symptoms, and the exclusion of compression of the cord (page 882), ataxic paraplegia (page 858), subacute combined (toxic) degeneration of the cord (page 858), disseminated sclerosis (page 859), and meningitis (page 828).

Prognosis.—The acute cases often soon lead to death; this is almost inevitable in transverse cervical myelitis. In other cases more or less improvement may ensue, but the patient usually becomes permanently incapacitated. Death may result from secondary infections, such as pneumonia, cystitis, proctitis, bed sores, etc.

Treatment.—Rest in bed is essential, but the patient should be frequently moved from side to side and on his back, and the most painstaking care must be exercised to prevent bed sores, cystitis, etc. The bladder must be regularly evacuated by catheterization, and the rectum by the use of laxatives and enemas. During the active stage of the disease good results have been claimed from the use of the salicylates, hexamethylenamin, and mercury (by inunction), even in non-syphilitic

cases. Painful contractures of the muscles may be relieved by massage, hot applications, the bromides, etc. After the subsidence of the acute phenomena, strychnine, massage, electricity, warm baths, etc., are indicated, and orthopedic and other surgical interventions to prevent and correct deformities.

COMPRESSION OF THE SPINAL CORD.

(*Compression Myelitis.*)

Etiology.—Compression of the spinal cord may result from caries of the vertebræ (usually tuberculous, sometimes syphilitic), fracture-dislocation or tumor of the vertebræ, tumor or cyst of the spinal meninges, and aneurysm of the thoracic or abdominal aorta.

Pathology.—In the case of fracture or dislocation the compression may occur suddenly, and is attended by varying degrees of crushing up to complete destruction of a segment or severance of the cord, with associated hemorrhage. In other cases the compression is slow, though usually progressive; the cord where compressed is smaller and paler than elsewhere, and reveals more or less secondary degeneration of its various tracts and replacement neuroglial fibrosis. Various grades of meningitis may be associated, especially in the tuberculous and syphilitic cases.

Symptoms.—In the traumatic cases of sudden onset the symptoms vary with the grade of compression; they may be those of a total transverse lesion (page 837). In the cases of more insidious onset muscular twitchings and pains, radiating in the course of the nerves, may occur from irritation of the nerve roots. In the course of time various paralytic and sensory phenomena develop, varying with the seat of the compression; these in general are analogous to those mentioned in connection with myelitis (page 880) and chronic hypertrophic pachymeningitis (page 828).

Diagnosis.—The diagnosis depends upon the symptoms mentioned and the signs of the primary disorder causing the compression. Special attention should be directed to detecting the early stages of caries of the vertebræ, which, because of the undefined character of its symptoms, often passes unnoticed: persistent pain (lumbago, etc.), vertebral tenderness, muscular rigidity along the spine, more or less immobility and difficulty in executing vertebral movements, etc., should not be overlooked. When kyphosis, skoliosis, etc., have developed, the diagnosis is self-apparent.

Prognosis.—The prognosis depends upon the cause, degree, and duration of the compression.

Treatment.—The treatment is that of the primary cause. In tuberculous caries marked benefit and cure may be effected by rest, various orthopedic apparatus, and the general treatment applicable in tuberculosis; the extent of the improvement in the nervous symptoms will

depend upon the amount of permanent damage done to the cord. Syphilitic cases require antisyphilitic treatment. Cases due to fracture, dislocation, or tumor may be relieved by surgical measures (laminectomy, etc.).

TUMORS OF THE SPINAL CORD.

Etiology.—Tumors of the spinal cord are more common in males than in females, and they may occur at any age, although they are most common in young adults. The etiological factors are those of tumors in general and are as ill understood.

Pathology.—Tumors of the spinal cord may be primary or secondary, and benign (so-called) or malignant; and for practical purposes, as in the brain, gummas and large or solitary tubercles are included among the tumors of the spinal cord. These spinal-cord tumors may be divided into: (1) The medullary or intramedullary growths (those within the substance of the cord). These comprise not more than 20 to 25 per cent. of spinal tumors; the most common are the gumma, the tuberculoma, and the glioma (gliosarcoma). They give rise to enlargement and distortion of the cord at the seat of growth, and to various secondary degenerations of the tracts destroyed or compressed. The glioma may become the seat of secondary softening and cavity formation (see syringomyelia, page 884). (2) The extramedullary growths, which develop outside the cord—from the spinal membranes, intravertebral fibro-alveolar tissue, the vertebræ, the intervertebral cartilages, etc., and comprise endotheliomas, sarcomas, fibromas, lipomas, hydatid and other cysts, gummas, tubercles, and secondary carcinomas. These compress the cord and give rise to lesions analogous to those due to compression from other causes (page 882). Tumors in general may develop in any part of the cord; they are more common on the dorsal and lateral than on the ventral surface.

Symptoms.—*Intramedullary tumors* give rise to initial motor paralysis and sensory disturbances, varying with the seat of development. Occasionally there is an early localized muscular atrophy (destruction of the cells in the anterior cornua); and this may be combined with dissociated anesthesia. In other cases the symptoms are those of a unilateral lesion (Brown-Séquard paralysis, page 838); that is, chiefly motor paralysis on the side of and below the segmental level of the lesion, and anesthesia on the other side. The tumor may continue to grow and ultimately give rise to symptoms of a more or less complete transverse lesion (page 837). In other cases, in which the gray matter is especially involved, the symptoms of more or less outspoken syringomyelia ensue.

Extramedullary growths give rise to pain, due to compression of the dorsal roots; generally at first this is unilateral, and it radiates variously, although in "root distribution" depending upon the segmental level of the lesion. Irritation of the anterior roots may cause muscular

twitchings and rigidity. Subsequently, in consequence of compression of the cord, various motor (paralytic) and sensory disturbances ensue. These at first may be of the Brown-Séquard type; later, when there is more or less complete transverse compression of the cord, the symptoms consist chiefly of paralysis and wasting of the muscles corresponding to the segmental level of the growth, and anesthesia corresponding to the dorsal roots compressed; spastic paralysis without wasting below the level of the lesion; and perhaps later more or less complete anesthesia below the lesion and loss of control over the vesical and rectal sphincters.

Diagnosis.—The diagnosis is often in doubt; but when present, pain radiating in a root distribution and progressive paralysis are almost conclusive evidence of an extramedullary growth. In other cases localized muscular atrophy and associated anesthesia, with the later development of Brown-Séquard paralysis or paraplegia, indicate an intramedullary growth. In most cases, however, one must be content with the diagnosis of intraspinal tumor, and should determine the level of the growth by attention to the segmental distribution of the motor and sensory phenomena. One must exclude compression of the spinal cord from other causes (page 882).

Prognosis.—The outlook is bad unless the growth be a gumma or amenable to surgical removal, which, of course, is impossible in intramedullary growths.

Treatment.—The treatment is wholly surgical, unless the growth be a gumma. In case of doubt, therefore, recourse should be had to a course of antisyphilitic treatment for four to six weeks. If improvement does not follow, operation should be undertaken; otherwise, the treatment is symptomatic.

SYRINGOMYELIA.

(*Spinal Gliosis.*)

Syringomyelia is a condition characterized by neurogliomatous new-growth about the central canal, with subsequent cavity formation extending a variable length throughout the spinal cord. It should be distinguished from hydromyelia or dilatation of the central canal, which may occur, usually in minor grade, as a congenital condition or in consequence of the pressure of tumors, etc.

Etiology.—Syringomyelia occurs especially in early adult life, and in both sexes. The definite etiological factors are not known.

Pathology.—The cavity formation is most common in the cervical and upper thoracic portion, although it may extend throughout more or less of the length of the cord; it varies much in shape, being usually more or less rounded, but it may extend into one or both dorsal cornua; it generally takes the place of the central canal, but it may develop about the central canal, which may be found compressed somewhere in the periphery. The

condition is commonly believed to be the result of primary proliferation of the neuroglial tissue with consecutive degeneration and cavity formation. Some observers attribute it to a congenital defect of development, with distention of the central canal (hydromyelia) and consequent proliferation of the surrounding neuroglia. Although this condition may occur, syringomyelia seems undoubtedly to be a primary neurogliosis; the epithelial lining of the cavity found in some cases is that of the original central canal.

Symptoms.—The symptoms are of slow and insidious onset and progression; they vary with the position, size, and direction of the neurogliosis and cavity formation; and they result from destruction of the spinal-cord substance at different segmental levels, and from pressure upon adjacent tracts in the cord. Since the gray matter is primarily and most extensively involved, the following characteristic signs occur: (1) Dissociated anesthesia—that is, loss of the pain and temperature (protopathic) sensibility, with good or complete preservation of tactile (epicritic) sensibility in any part of the body; (2) progressive muscular atrophy, with fibrillary twitchings, and ultimate reactions of degeneration, usually developing first and remaining most marked in the arms (destruction of the cells of the lower motor neuron); and (3) trophic and vasomotor disturbances of the skin, such as injuries and burns not felt (loss of pain sense), local hyperemias and anemias, excessive sweating or dryness, localized atrophy, bullæ, painless whitlows and abscesses, gangrene, perforating ulcers, etc.; and of the bones and joints, such as arthropathies (considerable effusion into the joint, thickening of all the joint tissues, with final absorption of the bone and atrophy of the articulating surfaces); these resemble the similar arthropathies of tabes dorsalis, but are more common in the shoulder, elbow, and wrist than in the knee or ankle. Spontaneous painless fractures also may occur (due to brittleness of the bone). In addition, there may be more or less spastic paraplegia (involvement of the pyramidal tracts), or the manifestations of more or less complete transverse myelitis (page 880), depending upon the extent of the horizontal involvement of the cord. Often there are symptoms of unilateral (or perhaps bilateral) paralysis of the cervical sympathetic nerves (page 824), from involvement of the first thoracic segment (spinal centre for the cervical sympathetic). Extension of the lesions to the medulla may give rise to atrophy and fibrillary twitchings of the muscles of the tongue and face (usually, at first at least, unilateral), dissociated anesthesia of the face and head, oculomotor paralysis and nystagmus; and difficulty in speaking, swallowing, and breathing, and cardiac arrhythmia (so-called syringobulbia). Progression of the lesions to the sacral cord may result in loss of control over the vesical and rectal sphincters.

Morvan's disease, a condition characterized by painless whitlows and other trophic or nutritional disorders of the hands, such as ulcerations, necroses, falling of the nails, etc., is now believed to be a variety of syringomyelia. The cases studied of recent years reveal also muscular

atrophy and dissociated anesthesia, as well as cavitation of the spinal cord (when studied after death).

Diagnosis.—The disease may be overlooked in the early stages, unless the dissociated anesthesia is carefully sought for; but this, the progressive muscular atrophy, and the trophic disorders make the diagnosis easy.

Prognosis.—The disorder, as a rule, is slowly progressive, but it may last many years, and sometimes comes to a standstill; it is not in itself dangerous to life unless the medulla becomes involved.

Treatment.—The treatment is purely symptomatic—massage and electricity to keep up the nutrition of the muscle, and perhaps strychnine as a general tonic.

NERVOUS DISEASES OF FUNCTIONAL OR UNKNOWN NATURE.

MIGRAINE.

(*Hemicrania; Sick Headache; Bilious Headache.*)

Migraine is a disorder characterized by paroxysmal attacks of severe headache, nausea, vomiting, and prostration, and usually preceded by certain visual disturbances.

Etiology.—Migraine is much more common in women than in men; it occurs in neurotic subjects; and it sometimes, but not always, exhibits hereditary or familial tendencies. In the great majority of cases it is a reflex manifestation of disease of some organ of the body, and is found especially related to errors of refraction, disease of the naso-pharynx, of the female genitalia (especially dysmenorrhœa), and of the gastro-intestinal tract; in some cases it seems to be autotoxic and a manifestation of gastro-intestinal auto-intoxication, gout, uremia, etc.; it is perhaps sometimes vasomotor in origin—vasoconstriction followed by vasodilatation of the cerebral vessels and headache (suggested also by a primary facial pallor and blueness and coldness of the extremities and secondary flushing and perhaps sweating); it may replace an epileptic attack; in some cases no adequate cause can be assigned. The attacks may be induced by excessive and prolonged eyestrain (week-ends in school children and other students), by general fatigue, dietetic indiscretions, constipation, menstruation, emotional excitement, etc.

Symptoms.—In the majority of cases there are certain premonitory symptoms (for hours, a day, or more) that enable the patient to foretell the advent of an attack; these are especially visual—flashes of light, colored spectra, dimness of vision, etc.; or the patient may feel generally

dull, "headachy," and somnolent. The onset may be sudden, or there may be a more gradual increment of the premonitory headache; at all events, the headache becomes severe, and although usually limited to one side or temple (hemicrania), it may be bilateral and general. It is increased by auditory, visual, and sensory impressions of all kinds. In an hour or thereabouts nausea and vomiting supervene; retching may be marked, and, with the headache, leads to marked prostration. In the course of a short time the patient usually falls asleep, and awakes several hours later much refreshed, if not entirely well; in some cases, however, the attack may last two or three days. Various sensory phenomena are sometimes associated, such as numbness and tingling or anesthesia of the limbs; sometimes transient muscular weakness, or vertigo, tinnitus aurium, deafness, aphasia, etc. The attacks may occur at long or short intervals, and may continue throughout life. In women they may cease about the time of the menopause, and at about the age of fifty years in men.

Diagnosis.—Although migraine is typically paroxysmal and unilateral, and attended by visual and other sensory disturbances, and nausea and vomiting, all attacks are not typical; other causes of headache, therefore, must be inquired into—all possible sources of reflex irritation; diseases of metabolism, of the blood, of the cardiovascular system, of the urinary system, and of the brain and its membranes and the skull. In neuralgia there are paroxysms of pain, radiating in the area of distribution of a nerve, and tender spots along the course of the nerve; and the visual signs and the nausea and vomiting of migraine do not occur.

Prognosis.—The prognosis is the better the earlier the patient is thoroughly studied and all possible sources of reflex irritation eliminated. The outlook is bad for cure in cases of long standing, but in even the most intractable cases much relief can be afforded the patient.

Treatment.—The most essential part of the treatment is to prevent the attacks. In the great majority of cases this can be done by the proper correction of errors of refraction—even slight errors; in other cases, other sources of reflex irritation must be sought and adequately treated; the diet should be regulated (purin-free diet), the bowels opened thoroughly daily, a hygienic mode of life insisted upon, and all factors that in any way tend in the individual patient to provoke an attack corrected. Bromides, salicylates, sodium benzoate, and potassium acetate (and other alkalies) also are serviceable in preventing attacks; as are also the saline cathartics taken daily before breakfast. During the attack the patient should go to bed in a quiet, darkened room, and abstain from food. If retching continues, it may be best relieved by lavage; local hot applications, bismuth, cerium oxalate, cocaine, etc., may also be tried. The bowels should be opened by calomel or blue mass and a saline cathartic. Acetphenetidin, acetanilide, and similar drugs are often necessary for the headache; or one may give caffeine, sodium benzoate, or nitroglycerin (in the event of high blood pressure), or gelsemium or cannabis indica.

EPILEPSY.

Epilepsy is a disorder of the nervous system, characterized by recurring attacks of loss of consciousness, attended or not by convulsions. The attacks of momentary loss of consciousness unattended by convulsions are know as *petit mal*, or slight attacks; those in which there is loss of consciousness and general convulsions are known as *grand mal*, or severe attacks; and those in which the convulsive seizures remain localized to a certain definite area or group of muscles, or at least always begin therein, and are often unattended by loss of consciousness, are known as Jacksonian epilepsy.

Etiology.—In a majority of the cases epilepsy begins before puberty, and it involves the sexes equally. It rarely develops *de novo* after the twentieth year, the cases observed thereafter being usually either recurrences of attacks occurring for the first time during childhood, or Jacksonian epilepsy due to a local cortical lesion. In the so-called idiopathic epilepsy no definite cause can be assigned, although etiological importance is attributed to certain hereditary influences, such as epilepsy, insanity, hysteria and other neurotic conditions, syphilis, and chronic alcoholism in one or both parents. In other cases the disease, or at least epileptiform convulsions, may be due to: (1) Injuries to the head received during birth, or vascular (hemorrhagic) disorders suffered soon thereafter and followed by persisting residual lesions (cerebral paralyses of infants and children). (2) Toxemias, such as infectious diseases (in infants and children), alcoholism (especially common in middle-aged and elderly chronic alcoholic subjects), plumbism, uremia, intestinal autointoxication (especially in infants and children). (3) Reflex factors, such as dentition, an adherent prepuce, intestinal worms, a foreign body in the auditory canal or the nose, errors of refraction, the irritation produced by a cicatrix, onset of puberty, menstruation, etc. (4) Cardiovascular disease, such as arteriosclerosis, which sometimes causes convulsive seizures in elderly subjects, and if associated with disorder of the bundle of His may lead to Adams-Stokes disease (page 420). (5) Organic disease of the brain, especially fracture of the skull and resulting irritation of the brain by a spicule of bone, local meningitis, etc.; brain tumor; brain abscess; syphilis, etc. Many of these conditions can scarcely be classed as epilepsy in a true sense, although the convulsive seizure may be indistinguishable from that of true epilepsy; some of them are promptly cured by the removal of the exciting cause of the attack, such as a foreign body in the auditory canal, an adherent prepuce, a spicule of bone irritating the cerebral cortex, etc.; but in some of these cases the "epileptic habit" may have become acquired, and the convulsive seizures may not subside for some time.

Pathology.—In the idiopathic cases there is no recognizable lesion. In the other cases there are the various lesions peculiar to the disorders mentioned.

The epileptic fit may be looked upon as a sudden discharge of nervous energy, provoked by influences which may be: (1) Toxic in nature, such as the poison of uremia; the products of perverted metabolism, such as ammonium carbamate, etc.; (2) irritative, such as the various organic disorders of the brain mentioned; or (3) vascular; and these various toxic and other stimuli may, if sufficiently active, provoke the discharge of nervous energy (the convulsion) by acting upon normal nerve cells; but, as is likely more common, being not excessively active, they unduly stimulate subnormal or highly sensitive cells and provoke an altogether disproportionate discharge of nervous energy.

Symptoms.—Grand Mal.—A typical attack of *grand mal* consists of an aura, a convulsive seizure with loss of consciousness, and certain after-effects. The attack apparently may originate in any part of the cerebral cortex, and the aura or premonitory sensations vary accordingly. These may be psychical in nature (a vague, dreamy, or terrified state); associated with the special senses—visual (occiptal lobe), auditory (temporo-sphenoidal lobe), olfactory or gustatory; related to the abdomen and the viscera (sensations of epigastric "sinking" or distress, or palpitation or other sensations related to the heart—pneumogastric aura); or there may be a sensation of numbness or other paresthesia in a finger or a hand (sensory aura), or localized twitching (motor aura). These auræ may be absent; when present, they are usually of momentary duration —a few seconds to perhaps a minute or thereabouts. Very soon the patient emits a loud scream or yell (epileptic cry), falls to the ground without any conscious effort to protect himself from injury, and is immediately in a convulsive seizure and unconscious. The spasms, which may begin anywhere, soon become generalized, although not everywhere equally marked. At first they are tonic, and involving the muscles of respiration, lead to cyanosis and venous distention; in a few seconds they become clonic, intermittent, jerky, and often violent, provoking marked tossing about of the patient's limbs and body; the eyelids open and close, the eyes move about in varying directions, the face is intermittently distorted and relaxed, and the jaws open and close violently, in consequence of which the tongue is usually lacerated and blood-stained saliva issues from the mouth. The urine often and the feces sometimes are discharged involuntarily; and petechial hemorrhages may occur into the skin and conjunctivæ. As a rule, in the course of one or two, rarely three or more, minutes the convulsive movements gradually cease and the patient, fully relaxed, falls into a profound coma. In some cases he can be more or less aroused after a short time, but he may sleep for hours and awake mentally confused and complaining of headache and muscular soreness; and there may be other after-effects, such as a trance-like state, loss of memory, aphasia, monoplegia, hemiplegia, mania, etc.—all of which soon subside, as a rule. Or he may become subject to post-epileptic automatism, and perform apparently purposeful acts, such as walking, riding, journeying, criminal acts, etc., without subsequent recollection thereof. In some cases the period of sleep is very short—rarely

perhaps absent—so that soon after even a rather severe convulsion the patient may return to his former occupation. Attacks may occur once or more frequently daily, or at longer intervals—weeks, months, years. In some cases a number of convulsive seizures follow in rapid succession, the patient meanwhile remaining entirely unconscious; this, so-called *status epilepticus*, may continue for hours or a day or more; it is often attended by fever and rapid pulse, and may soon lead to death from exhaustion.

Petit Mal.—Attacks of *petit mal* consist of momentary loss of consciousness, perhaps also vertigo and faintness, but no convulsions. The patient may suddenly stop talking or performing some act, drop what he may hold in his hand, or take up some new act, and almost immediately revert to his former activity. The attack may pass almost, if not quite, unnoticed by the patient's associates, or they may observe a transient pallor of the face or twitching of a muscle. In other cases the patient may experience only the aura of the more severe *grand mal*. These slight attacks may occur alone at long or short intervals, or they may alternate with severe seizures.

Jacksonian (Cortical, Partial) Epilepsy.—Jacksonian epilepsy is characterized by convulsive seizures that begin and usually remain localized in a certain definite group of muscles, and are often unattended by loss of consciousness; but in some cases the convulsions become more widespread, and consciousness may finally become lost. A study of the beginning of the convulsive seizures, commonly in the finger, hand, toe, or face, may lead to an accurate localization of the lesion, which is commonly an irritative lesion of the motor or sensory cortex.

Diagnosis.—*Grand mal* is usually readily recognized by the aura, the epileptic cry, the sudden loss of consciousness, tonic and later clonic spasms, laceration of the tongue, involuntary voiding of the urine, the postconvulsive sleep, and other after-effects. Attacks often occur at night, and, if unobserved by others, may be recognized by the involuntary voiding of urine, laceration of the tongue, and a rather common mental confusion and headache in the morning. Hysterical attacks may be distinguished by associated emotional disturbances and hysterical stigmas; usually a gradual onset and rarely an injury when falling; crying, talking, or screaming during the attack; absence of complete unconsciousness, and of lacerations of the tongue and voiding the urine during the attack; longer duration of the attack, and the frequent possibility of stopping it by procedures such as throwing water in the patient's face, etc. Attacks of *petit mal* may be distinguished from vertigo due to other causes and syncope by the occurrence of actual, although momentary, loss of consciousness, and the absence of the ordinary causes of vertigo and syncope (heart disease, Ménière's disease, etc.). Jacksonian epilepsy is in itself characteristic. The epileptiform convulsions due to other mentioned etiological factors should be recognized by determining the presence of those causes in the individual case.

Prognosis.—The prognosis varies, and depends upon the age when the disease developed, its severity, and the possibility of removing exciting

causes of the attack. In a considerable proportion of the cases cessation of the attacks or cure can be effected, but a tendency sometimes persists for the attacks to recur in later life. The prognosis is poor in the cases beginning in children and after the thirty-fifth year (many syphilitic); it is relatively good in those developing about puberty. In many cases, especially in cases of frequently recurring attacks, the mentality gradually becomes impaired.

Treatment.—In all cases, upon the development of fits the most painstaking care must be exercised to discover any possible source of irritation, since in the early stages removal of local irritations may lead to mitigation if not cure of the disease. The best possible hygienic conditions must be secured. The child should not be forced to keep up in studies with others better endowed. Excellent results have been achieved at epileptic colonies where the patients are taught some interesting and useful manual occupation. The diet should be light, nutritious, and nonstimulating; recent studies suggest the wisdom of reducing foods containing purin bodies (meat, fish, tea, coffee, etc.), and some observers believe in the desirability of reducing the sodium chloride intake. The bowels should be opened regularly and thoroughly; colonic flushings seem sometimes to be of service (lessening intestinal auto-intoxication?).

The epileptic seizure requires no special treatment, aside from care to prevent the patient's injuring himself; a mouth gag or similar contrivance between the teeth to prevent his biting his tongue (usually impossible, on account of the brevity of the attack); and loosening the clothing to facilitate breathing.

On the whole the best medicinal treatment still seems to consist of the use of the bromides, despite some opinions to the contrary. Any of the bromides may be given, although perhaps the mixed bromides secure the best results. They may be combined with antipyrin (5 grains, 0.3 gram, thrice daily—H. C. Wood); or, to counteract the tendency to produce acne, they may be given as follows:

℞—Potassium bromide	4 drams	16	0
Sodium bromide,			
Ammonium bromide, of each	2 drams	8	0
Solution of potassium arsenite	45 minims	3	0
Camphor water, sufficient to make	8 ounces	250	0—M.

S.—Two teaspoonfuls (10 c.c.) in water three times daily.

Strontium bromide also may be used; and the dose of the bromides in general may be varied to suit the individual patient; more than 40 to 50 grains (2.5 to 3.25 grams) daily is rarely required. The cases not benefited by these doses are not likely to be benefited by larger doses, and large doses not only produce acne, but almost certainly cause mental depression (bromism), and sometimes ultimate mental deterioration. Belladonna, atropine, sodium borate, nitroglycerin, thyroid extract, and other drugs, alone or in combination with the bromides, have also been recommended. In status epilepticus the bromides, chloral, morphine, hyoscine, etc., are indicated, and stimulants if the patient goes into

collapse. In Jacksonian epilepsy operation is often of the greatest utility; it should be undertaken if the focal symptoms are definite and referable to an accessible region of the brain, especially if there is a history of injury to, or a depression of, the skull.

HYSTERIA.

Hysteria is one of the so-called functional disorders of the nervous system, consisting of a permanent abnormal psychic state (hysterical temperament), abnormal suggestibility, and divers transient motor, sensory, vasomotor, trophic, and other neurotic phenomena.

Etiology.—Hysteria is very common in females, but it occurs also in males—more commonly than is generally supposed—and it is not uncommon in children. Its manifestations may become obtrusive at any age, but they are most common at or soon after puberty or in early adult life. Heredity—hysteria, epilepsy, insanity, neurotic instability, etc., in one or both parents—is of predisposing influence; almost equally important is bad early training and education. The exciting causes of the initial manifestations comprise traumas, shocks of various kinds, emotional disturbances, etc.

Pathology.—No definite lesions are known; the disease, therefore, is called functional, although doubtless there is some lesion which still eludes detection. Hysterical phenomena, of course, may accompany organic disorders.

Symptoms.—The basis of hysteria is the permanent hysterical temperament, which is said by Starr to be manifested "by an abnormally keen sensibility to all external impressions and sensations, by a high grade of imaginative power, by a susceptibility to suggestions, by an unusual desire for attention and notice, by variations in mood not due to apparent causes, by a lack of judgment, by a manifest incapacity to exercise control over thought, emotion, and action, and by a tendency to act on sudden impulses."

The motor phenomena consist of convulsive attacks, paralyses, spasms, and tremors. The convulsive attacks (hysteria major), which are rather uncommon, bear some resemblance to epilepsy, which has suggested the term hystero-epilepsy—a misnomer. They are usually preceded by emotional disturbances, globus hystericus (a sensation of a lump or obstruction in the throat and consequent suffocation), etc. The onset is usually rather gradual, although it may be sudden; consciousness is not wholly lost; a tonic stage is followed by clonic movements and the adoption of various more or less grotesque attitudes. The attack often lasts upward of a half hour or more, and may be succeeded by a period of mental aberration—pseudomania, a dreamy state, lethargy, trance, etc. Incomplete or abortive attacks (hysteria minor) may occur; they are unassociated with convulsive seizures and may consist of attacks of causeless laughing and crying, grotesque attitudes, passionate attitudes (ecstasy),

catalepsy, somnambulism, etc. Hysterical paralysis may be monoplegic, hemiplegic, paraplegic (most common), or affect any or all the limbs variously, or only groups of muscles. The paralyses may come on slowly or suddenly and they may disappear as they came; they may be partial or complete, and they are often preceded by more or less muscular weakness (hysterical myasthenia). Some muscular atrophy may follow (from disuse), but there are no reactions of degeneration; the knee-jerk may be normal or exaggerated (never absent); an imperfect ankle clonus is said to be occasionally observed, but the Babinski sign is never elicited. The superficial reflexes may be abolished, especially when sensory changes are associated. Control over the vesical and rectal sphincters is maintained, but retention of the urine, with overflow, may occur. Partial or complete aphonia may ensue from paralysis of the laryngeal muscles, usually the adductors. In some cases of paraplegia the loss of power is apparent, not in the reclining or sitting posture, but only when the patient attempts to stand (astasia) or walk (abasia); in these cases there is usually no ataxia and no disturbances of sensation; most, if not all, of the cases are hysterical. There may be also involvement of the pharyngeal and other muscles supplied by the cranial nerves. Muscular spasms often occur; involving the arm, leg, or neck, they give rise to various contractures and deformities (which may relax during sleep or anesthetization); involving the muscles supplied by the cranial nerves, they may cause spasmodic ptosis, strabismus, deviation of the tongue, spasmodic œsophageal obstruction, etc. Sometimes peculiar dancing movements occur (a form of saltatory spasm). Tremors are very common; they may be fine or coarse, rhythmic or arrhythmic, choreiform; and they may involve any part of the body, especially the eyelids, mouth, tongue, fingers, shoulders, legs, etc.

The sensory phenomena consist of anesthesia, hyperesthesia, and paresthesia. Anesthesia is the most common; it is usually a hemianesthesia (skin and mucous membranes), which may be partial or complete for all types of sensibility, or the sensibility may be dissociated; in other cases it is limited to a distal portion of a limb (glove or stocking form), or a segment (cuff form); or it may occur in variously shaped patches, which may change from time to time. Hyperesthesia is a significant symptom, the basis of so-called hysterogenetic zones; these are most common over the ovaries, beneath the breasts, just below the ribs, and over the vertebræ (see Fig. 15, page 804). Pressure over these tender spots may induce hysterical "attacks," and promote their cessation when they have developed. Paresthesic phenomena of various kinds (sensations of heat and cold, numbness, tingling, etc.) may be observed variously in different cases. Sensory phenomena referable to the special senses, especially the eyes and ears, are common and significant. The most important consists of concentric contraction of the field of vision, usually bilateral, but often more marked on the side of hemianesthesia (crossed amblyopia); perversion of color perception (dyschromatopsia), consisting usually of the recognition first of red or green, instead of the

normal sequence of blue, yellow, red, green, and violet; sometimes all color perception is lost, everything appearing dull or grayish (achromatopsia). Hysterical deafness may occur, and the sense of smell and of taste may be impaired or lost, usually only or more markedly on the side of hemianesthesia; and there may be also a nervous hysterical anorexia, vomiting, constipation, etc.

Vasomotor and trophic phenomena are often observed in varying combinations with the other phenomena; they consist of erythematous eruptions, œdema, urticaria, dermographism, cutaneous gangrene (neurotic or hysterical dermatitis), etc. Fever (doubtless of artificial production) may be observed; and disorders of metabolism and of the secretions and excretions may occur.

Diagnosis.—Hysteria may simulate almost, if not quite, all other nervous diseases; but, as a rule, it can be more or less readily distinguished, because the clinical picture is not typical of the simulated disease, and because, in addition, hysteria itself manifests certain characteristic phenomena. Hysterical phenomena are characterized by their frequent sudden onset, variability from day to day, and frequent sudden cessation; by the character of the motor paralyses, and their association with the anesthetic and other so-called stigmas of hysteria; and by their occurrence in a person of hysterical temperament. The differentiation of the attacks of major hysteria from epilepsy is mentioned on page 890.

Prognosis.—The prognosis is good; many patients recover entirely spontaneously, and others under well-directed management. Many, however, may readily be made worse by a tactless physician; indeed, Babinski and other observers believe that almost, if not quite, the whole complex of hysteria (the motor, sensory, and other phenomena) results from suggestion to a susceptible subject by a maladroit or unskilful physician or other person: assuredly an extreme and not proved view.

Treatment.—The treatment should consist in improvement in the general health, the eradication of any functional defects aside from the purely hysterical, good food, fresh air, etc. In addition, the hysterical temperament and the abnormal suggestibility of the patient must be borne in mind—its potentialities for good as well as evil. The successful physician will exercise a firm, although tactful, control over the life and activities of the patient—for a time at least, regulating and commanding the daily life and diet of one; preventing introspection in another by enlisting her interest in some useful and absorbing occupation, charitable work, outdoor exercise, etc.; prescribing a modified, complete or incomplete Weir Mitchell rest cure in another, or a work cure in another, or a course of massage, electricity, or hydrotherapy. A resourceful psychotherapy finds here a special field of usefulness. In addition, tonics, such as iron and arsenic, and nervines, such as valerian and asafœtida, are useful from time to time. Hypnotism is rarely if ever indicated, and assuredly never in inexpert hands; it is a two-edged sword, and often cuts in the adverse direction.

NEURASTHENIA AND PSYCHASTHENIA.

(*Nervous Debility or Disability; Nervous Exhaustion; Nervous Prostration.*)

Neurasthenia is a disorder of the nervous system, unassociated with demonstrable anatomical lesions, and manifested clinically by marked mental and bodily fatigue, which upon effort develop rapidly and disproportionately to the exciting cause; by undue irritability and response to external stimuli; and often by disturbances of the emotional and psychic functions. Psychasthenia nowadays is sometimes used as a synonym for neurasthenia, and it is sometimes restricted to that type of neurasthenia in which the symptoms are mental rather than spinal.

Etiology.—Neurasthenia may occur in men and in women. In a great majority of the cases there is a predisposing congenital factor—a nervous system ill adapted to the stress and stain (mental, physical, emotional) of modern life—well borne by the majority of mankind. One or both parents of neurasthenic or psychasthenic subjects are often found to have been neurasthenic, epileptic, hysterical, excessively emotional, alcoholic, insane, etc., and they transmit an unstable nervous system. Determining factors of the nervous breakdown comprise overwork (physical and mental), worry, misdirected education, shocks, anxiety, insomnia, excessive alcoholism and venery; infections, such as typhoid fever, syphilis, etc.; auto-intoxications, such as chronic gastric and intestinal derangements, gout, diabetes, nephritis, etc.; exogenous intoxications, such as ptomaine poisoning, plumbism, mercurial poisoning, etc.; disease of various organs of the body, such as the female genitalia, the kidney (nephroptosis), stomach (gastroptosis), eyes (eyestrain), etc.; prolonged residence in the tropics; and trauma. Most of these determining factors do not cause neurasthenia in the absence of the congenitally weak or unstable nervous system—the primary factor.

Symptoms.—The symptoms differ in different cases, and they comprise a variable congeries of mental, motor, sensory, vasomotor, somatic, and sexual symptoms. The mental symptoms are almost always present and usually dominate the clinical picture (psychasthenia); they consist of general "nervousness," inability to concentrate the attention or to persist in mental effort, which of itself may induce headache, vertigo, and mental confusion; imperfect reception and retention of perceptions, and consequent loss memory; loss of confidence in one's ability, delay and disinclination in coming to conclusions, and impaired judgment; morbid fears (agoraphobia, cleithrophobia), and more or less compulsory ideas (which, however, verge upon, if they do not constitute, insanity); irritability of temper and exaggerated reaction to external impressions —of sensibility, sight (retinal hyperesthesia), hearing (auditory hyperesthesia), smell, etc.; and emotional disturbances. The eyes are readily tired by reading or other "close work" (refractive errors should be searched for); the visual fields may be contracted, or may become contracted under examination (fatigue contraction). The motor

symptoms consist of muscular fatigue, readily induced and disproportionate to the exertion; tremors, especially of the hands, and twitchings or habit spasms, especially of the face; and usually normal or exaggerated tendon reflexes, which readily become exhausted. The sensory symptoms consist of a feeling of malaise, irregular, fugitive pains (in the head, the back, the legs, along the vertebræ), paresthesias (numbness, tingling, sensations of heat and cold), areas of hyperesthesia, especially about the head, along the vertebræ, etc. Vasomotor symptoms consist of readily induced pallor or flushing of the face, blueness and coldness of the extremities, excessive perspiration, dermographism, etc. The somatic symptoms comprise various symptoms referable to the heart (palpitation, vertigo, distressing pulsation of the arteries, etc.), of the gastro-intestinal tract (gastric neurasthenia, page 621), of the kidneys (lessening or increase in the amount of urine, phosphaturia), etc. The sexual symptoms (sexual neurasthenia) comprise various paresthetic phenomena, nocturnal emissions, spermatorrhœa, imperfect erection, premature ejaculation, etc., in the male, and hyperesthetic and paresthetic phenomena unassociated with organic disease, frigidity, nocturnal involuntary organisms, etc., in the female. The importance of these is often unduly exaggerated.

Diagnosis.—The diagnosis rests upon the generally inherited neuropathic basis and the activity of some one of the many determining causes. But many organic diseases of the nervous system and of other organs may begin with "neurasthenic" symptoms, so that careful and repeated physical examination and prolonged observation may be required to exclude other disorders.

Traumatic neurasthenia (the traumatic neuroses and psychoses) is a disorder of the nervous system that follow injuries and shocks, and is unassociated with gross lesions of the nervous system and the enclosing bony framework; it is often spoken of as "railway brain" and "railway spine." The condition is more common after accidents in which the patient may not be notably injured, but suffers a "mental shock" in consequence of the suddenness of the accident or disquieting attending circumstances (railway wrecks, etc.), than it is after accidents in which the patient may be more or less severely injured. The nature of the disorder is not known: it has been attributed to cerebral and spinal concussion; to organic lesions of the brain so slight as to elude detection; to disturbances in the intimate make-up of the cerebral cells; to psychic as distinguished from cerebral disorder; and to malingering pure and simple. The symptoms in general do not differ from those of neurasthenia caused by other factors; only from the history of an injury can the physician distinguish definitely the traumatic from the non-traumatic cases. The traumatic cases, however, as a rule, are rather more acute in onset, although they may not develop for days or weeks after the injury; mental symptoms are markedly in the foreground; hysterical manifestations are often associated, especially paralyses (monoplegic, hemiplegic, paraplegic) and anesthesias; and traumatic lumbago (due doubtless to

twisting or torsion of the vertebræ or stretching or laceration of the lumbar muscle and ligaments) may also be present. The condition may last a long time, and may suddenly clear up entirely (sometimes very soon after the successful issue of a lawsuit); but mental deterioration, melancholia, etc., may ensue.

Prognosis.—The prognosis should be guarded, since neurasthenia usually occurs in a person with a more or less subnormal nervous system; the breakdown may be entirely recovered from, but the predisposition to a relapse is always present; so that, following recovery, the patient's life and work must be ordered well within his capabilities.

Treatment.—As in the treatment of hysteria, so also in neurasthenia, success depends largely upon the physician, the confidence that he inspires in the patient (psychotherapy), and the attention that he gives to detail. All possibilities of organic disease should be excluded by examination and reëxamination of the patient and assuring him that none exists; if found, it should, of course, be treated appropriately. All the functions of the body, the secretions and excretions, the diet, the daily life, must be regulated in accordance with the exigencies of each patient; and all the possible factors determining the breakdown must be carefully sought and rigidly excluded from activity. Errors of refraction should be corrected; a modified, partial, or complete Weir Mitchell rest cure is of much benefit in selected cases; others are better adapted to a work cure, a change of scene and occupation, a prolonged sea voyage, etc. Hydrotherapy is one of the best therapeutic measures at our command. Electricity may form part of the exaggerated psychotherapy, the practice of which is now fashionable in certain quarters. The bromides for a short time are often serviceable, or one may use valerian, asafœtida, etc. Tonics, such as iron, quinine, nux vomica, arsenic, etc., are useful as occasion may demand.

OCCUPATION NEUROSES.

(*Professional Neurosis; Occupation or Professional Cramps.*)

The occupation neuroses comprise a series of disorders characterized by cramps of muscles or groups of muscles concerned in the frequent or prolonged execution of certain voluntary coördinated movements.

Etiology.—The occupation neuroses are more common in men than in women; most of the subjects are "neurotic," endowed with a nervous system ill adapted to the prolonged strain to which it is subjected. The cramps occur most commonly in writers (writer's cramp or palsy), but also in telegraphers, typewriters, seamstresses, pianists, and others; the movements ultimately leading to the cramp may be fine and complicated (such as writing), or coarse and more or less simple.

Pathology.—The nature of the disorder is not known; it has been variously ascribed to disorder of the muscles, the peripheral nerves, and the cerebral cortex; it is likely that much of the disorder emanates from the cells of the cerebral cortex, which perhaps become exhausted from

the necessarily prolonged effort to innervate the muscles; but whether this is the whole process is doubtful.

Symptoms.—The symptoms consist of the development of spasms in groups of muscles concerned in the special cccupational activity; the spasms, as a rule, are tonic; occasionally there is a clonic or jerky movement. The spasms develop only when the special occupational activity is undertaken, the same muscles or group of muscles contracting normally when acting independently or in other associations (in executing other movements). In writer's cramp there is a progressive disability; the movements of writing are performed with increasing effort and slowness; perhaps there is some associated tingling and aching in the muscles of the hand and the forearm, until finally writing becomes almost, if not quite, impossible from tonic spasm of the muscles; occasionally there may be a jerky clonic spasm (separation of the fingers, for instance), or tremors, or paresis. In a few cases some muscular atrophy, especially of the thenar, hypothenar, and interossei muscles, occurs (peripheral nerve disorder?). Similar phenomena, involving, however, other groups of muscles, occur in other circumstances, depending upon the occupation of the subject.

Diagnosis.—The disorder is readily recognized from the occurrence of the spasms only upon the patient attempting certain special acts, and from the ability to perform other acts involving the same muscles. Neuritis may be excluded by the absence of pain and tenderness along the course of the nerves and of muscular atrophy. In the event of tremor, other disorders attended by tremor, especially multiple sclerosis, paralysis agitans, and progressive paresis of the insane, must be excluded.

Prognosis.—Recovery may ensue if the patient discontinues the provoking occupation—at least for some time.

Treatment.—The first essential is discontinuance of the special activities that provoke the spasms. The muscles involved should also be protected from such fatigue as may follow prolonged muscular movements of other sorts. Locally, hydrotherapy and massage are often serviceable; perhaps also galvanism. Attention should also be paid to improving the general health, as in neurasthenia.

PARALYSIS AGITANS.

(*Shaking Palsy; Parkinson's Disease.*)

Paralysis agitans is a disease of the nervous system, unassociated with definite or constant anatomical lesions, and characterized clinically by muscular weakness, rigidity, and tremor, and a consequent characteristic facial expression, attitude, and gait.

Etiology.—Paralysis agitans usually begins after middle life and is twice as common in men as in women. The onset is variously attributed to mental shock, worry, trauma, etc., but their real significance is undeterminate.

Pathology.—There are no definite or constant anatomical lesions. Changes have been found in the cortical nerve cells of the cerebrum; in the

bloodvessels of the spinal cord (changes of an arteriosclerotic nature, and associated atrophic and sclerotic alterations in the gray and white matter of the cord); and in the muscles (especially in the muscle spindles). The disorder has been looked upon as a manifestation of senescence of the nervous system; as a toxemia; as the result of some disturbance of the parathyroid bodies, etc.

Symptoms.—The onset of the disease is insidious, although the first symptoms are sometimes referred to a shock or injury. The chief manifestation consists of a slowly progressive weakness and rigidity of the muscles, usually (but not always at first) associated with tremors. The tremors, which develop in at least two-thirds of the cases, are commonly first observed in the hands. In the beginning they may be slight and escape observation, but soon the forefinger and thumb (of one, but soon of both hands) are seen to be the seat of a so-called "pill-rolling" or "cigarette-rolling" movement; gradually all the fingers become involved; and ultimately the muscles of the arm, leg, trunk, and face may share in the tremor. The tremors are fine (four to seven to the second); they are present during rest and quiescence; they are diminished, sometimes abolished, at least temporarily, by voluntary movements; they usually cease during sleep, and they are increased by excitement. The really characteristic manifestation of the disease is the muscular rigidity, which develops insidiously and becomes universal. It gives rise to the characteristic fixed and expressionless facial features (Parkinson's mask); to the dull and monotonous voice (rigidity of the muscles of the lips, tongue, and larynx); to a characteristic attitude—arching of the head and body forward (stooping posture), and flexion of the arms and hands (general overcoming of the extensors and adductors by the flexors and abductors); to the characteristic gait—steps at first slow and shuffling, but soon becoming short and rather rapid (festination), due to forward propulsion of the body and advance of, and the patient's effort to catch up with, his centre of gravity; in consequence of this the patient may ultimately fall forward; if suddenly started or pushed backward or laterally, a corresponding retropulsion or lateral pulsion may be observed. This muscular rigidity also results in delay in initiating voluntary movements, and in resistance to passive movements; muscular spasms also may occur. Muscular power is usually relatively well maintained for a considerable time. The tendon reflexes may be normal or exaggerated. Subjective sensations of heat, sometimes of cold, and dull aching in the legs or arms may be complained of, but objective sensory changes do not occur. Vasomotor disorders, such as a dry skin, sweating or flushing, and increased surface temperature, are occasionally observed. The patient is often apathetic, but when interested his mental faculties are usually found normal; he perhaps does not think as slowly as he must express himself.

Diagnosis.—The disease is usually readily recognized; one must not overlook the cases without tremor. Multiple sclerosis is easily differentiated by the cessation of the tremor on voluntary movement (as

contrasted with the volitional tremor), and by the other characteristic signs (page 859). With advancing age, but usually not much before the seventieth year, a fine tremor often develops (*senile tremor*); it usually involves the hands, and sometimes the neck and head (causing a slight nodding movement) and the legs; but it is not associated with the other signs of paralysis agitans.

Prognosis.—The disease is incurable, although of itself it does not shorten life or lead directly to death.

Treatment.—The treatment comprises a quiet life, freedom from worry and distress, and the best hygienic and dietetic regimen. Hyoscine hydrobromate, duboisine, codeine and other preparations of opium, belladonna, cannabis indica, and extract of the parathyroid bodies have been recommended to control the tremors. Some benefit attends massage, hydrotherapy (hot baths), active and passive movements, and the use of tonics, such as arsenic, quinine, etc.

ACUTE CHOREA.

(*Sydenham's Chorea; Chorea Minor; Infectious or Rheumatic Chorea; St. Vitus' Dance.*)

Acute chorea is a disease almost certainly infectious, involving chiefly the nervous system, and characterized clinically by involuntary, irregular, jerky muscular contractions and purposeless movements, and frequently associated with endocarditis and various psychic disturbances.

Etiology.—Acute chorea is a disease of childhood, occurring especially between the fifth and the fifteenth year, and twice or thrice as commonly in girls as in boys. An inherited unstable nervous system (epilepsy, hysteria, insanity, and other neuropathic taints in one or both parents) seems to be a predisposing factor. The exciting cause appears to be an infectious agent, as yet undiscovered, but allied to, if not identical with, that of rheumatic fever; a history of rheumatism may be elicited in from 25 to 50 per cent. of cases of chorea, and cardiac murmurs are very commonly found clinically. Perhaps other infectious processes may be active etiologically; but in some cases a history or evidence of infection cannot be obtained. Fright, shocks, and divers forms of reflex irritation (worms, errors of refraction, the harass of too strenuous education, etc.), are often put down as exciting causes, but their influence is unquestionably overestimated.

Pathology.—No constant or characteristic lesions have been described. The disease is rarely fatal; but congestion, swelling, and degeneration of the cerebral cortical cells have been reported, and Diplococcus rheumaticus (Poynton and Payne) has been isolated from the meninges. The lesions of endocarditis are commonly associated.

Symptoms.—The onset of the disease is, as a rule, gradual. During what may be called a prodromal period, of a week or more, the child may be listless, inattentive, generally nervous, restless, and unable to

remain quiet. Soon, or in some cases, following a fright or emotional disturbance, the child is observed to be very nervous and to exhibit involuntary, irregular, jerky, and purposeless movements. These may involve one or both arms (most commonly the fingers), or the legs, the face, one-half of the body, or the whole body; they vary much in degree from those that are slight and may almost pass unobserved, to those that preclude the possibility of the child's remaining still, and if involving the facial muscles, result in characteristic grotesque grimaces; they are increased by excitement, and may be augmented in the hands and arm, for instance, by getting the child to attempt to grasp some object (associated incoördination); and they usually disappear during sleep. Involvement of the muscles of articulation results in defective speech, and sometimes various explosive (laryngeal) sounds; of the muscles of deglutition, in difficult swallowing; of the diaphragm and other muscles of respiration, in embarrassed, irregular, or spasmodic breathing; and the cardiac action may become irregular. There is usually at least some muscular weakness; at times this may become marked in one or more limbs (paretic chorea). The tendon jerks, usually difficult to determine, are normal or slightly exaggerated. Cardiac murmurs and other evidences of acute endocarditis (usually mitral) are present in more than one-half of the cases, and often persist after recovery from the chorea; perhaps in some cases the murmur is due to incoördinated action of the papillary muscles rather than endocarditis. Other rheumatic manifestations, such as joint disorders, erythema nodosum, subcutaneous rheumatic (sometimes called fibrous) nodules, etc., may be present. The child, as a rule, is fretful and unruly; sometimes more marked psychic symptoms, such as mental dulness or depression, hallucinations, delirium, mania, etc., may occur. At the end of two to four weeks the more acute symptoms usually tend to subside, but the attack commonly lasts from one to three months, and recurrences are not uncommon.

In some cases, however, the symptoms increase in intensity, or they may be very severe from the beginning—so-called *malignant chorea* (*chorea insaniens*). This is most common in early adult life and during pregnancy (toxic or infectious cases). The spasms are severe, general, and almost if not quite constant; they interfere with sleep and induce exhaustion; fever, delirium, and mania often ensue, and death usually results.

Diagnosis.—The diagnosis, as a rule, is easy. One must differentiate *habit chorea* (*habit spasm*; one type of convulsive tic), a disorder of children, especially neurotic girls, which may or may not develop out of the frequent repetition of volitional acts (mimicry), and which consists of rapid coördinated acts, usually of a purposeful nature, and partly or completely under the control of the patient's will. The movements are most commonly facial, such as winking, wrinkling the forehead, or drawing the mouth to one side, or they may consist of rapid shaking of the head, shrugging the shoulders, etc. The movements differ from those of acute chorea in their generally purposeful nature and in being confined to one

muscle or group of muscles (frequent repetition of the same movement). One must also exclude the athetoid and choreiform movements of the cerebral paralysis of children (history of the case, permanent paralyses, arrested mental development, etc.); hysteria (more regular movements and other stigmas of hysteria); and paramyoclonus multiplex (shock-like, often symmetrical muscular movements, frequently involving the trunk).

Prognosis.—The outlook is good, except in the so-called malignant cases. Recurrences, however, are common even in the mild cases.

Treatment.—The child must be removed from school, all disturbing and exciting influences must be guarded against, and all possible sources of reflex irritation must be inquired into, and, if found, corrected. The best results follow confinement to bed for some time, or at least to the quietude of a sunny, pleasant room. The diet should be nutritious and not stimulating; coffee and tea must be prohibited. Hydrotherapeutic measures (warm baths, cold packs, etc.,) and massage are often of the greatest service. Arsenic (solution of potassium arsenite) is of distinct value in most cases; it should be given in doses of 2 to 3 minims (0.12 to 0.2 c.c.) three times daily, and gradually increased to 6 to 10 minims (0.4 to 0.6 c.c.); larger doses should be avoided. Salicyl preparations, belladonna, cimicifuga, and other drugs have been recommended. Quinine, bitter tonics, cod-liver oil, are often useful adjuvants. The severe cases require the use of sedatives, such as the bromides, chloral, chloretone, hyoscine, cannabis indica, valerian, etc. The cardiac complications are to be treated as are similar conditions developing under other circumstances.

HEREDITARY CHOREA.

(*Huntington's Chorea; Chronic Progressive Chorea.*)

Huntington's chorea is a chronic hereditary disease of the nervous system characterized clinically by choreiform and ataxic movements, a staggering, ataxic gait, disorders of speech, and progressive mental deterioration. The disease bears no relationship whatever to acute (Sydenham's) chorea. Its nature has not been definitely determined; in the few cases that have come to necropsy chronic meningo-encephalitis, with atrophy of the cerebral cortex, especially of the frontal and parietal convolutions, has been found; in other cases, lesions analogous to hemorrhagic pachymeningitis. Some observers postulate a congenital defect or maldevelopment of the nervous system. The disease begins usually in adult life, after the thirty-fifth year, occurs in succeeding generations, and affects several members of the family—the males more frequently than the females. The movements begin in the hands and feet and finally involve the entire body, including the trunk; they resemble those of acute chorea in being jerky and purposeless, but they are usually slower and more incoördinate (ataxic), and they are likely to be

bilaterally symmetrical. The gait becomes ataxic and staggering, sometimes jerky; speech becomes slow and is sometimes varied by explosive sounds (involvement of the muscles of articulation). There is gradual mental deterioration, with loss of memory, delusions, etc., and final dementia. The disease lasts for years and is irremediable. It is readily recognized from its hereditary transmission, development in adult life, choreiform and ataxic movements, and progressive dementia.

CONVULSIVE TICS.

(*Tic Convulsif; Impulsive Tic; Gilles de la Tourette's Disease.*)

The convulsive tics comprise a group of disorders characterized by the more or less frequent repetition of involuntary spasmodic muscular movements of a generally purposeful nature. These tics are of cortical origin, and represent, therefore, a psychosis; they are, in many respects, hysterical in nature, and may be associated with other hysterical phenomena; they occur in neuropathic subjects, and usually develop in childhood, although they may come on in later life. Sometimes they develop out of habit spasms (page 901) and mimicry; in some cases they are perhaps of reflex nature, having developed in consequence of errors of refraction (blinking), of disease of the nose, mouth, etc. The disorder usually consists of involuntary, rapid, often lightning-like movements of the muscles of the face, such as blinking, wrinkling the forehead, twitching about the mouth, etc.; or there may be only fibrillary twitchings of certain muscles, such as of the eyelids (simple tics). In other cases the movements are more complicated, such as shrugging the shoulders, or convulsive movements of more or less of the body (complex tics). Sometimes the tics involving the laryngeal and associated muscles result in explosive cries or utterances; or words or names may be frequently repeated (echolalia), or obscene language used (coprolalia), or actions mimicked (echokinesis)—indicating the cortical nature of the disorder. A common psychic element in many cases is shown in the recurrence of a melody or harmony (mental equivalent of the muscular tic), in an almost irresistible impulse to touch objects (delire de toucher), to repeat words (onomatomania), to count before acting (arithmomania), in morbid fears (agoraphobia, cleithrophobia), doubts (folie de doute), etc. The disorder is usually readily recognized. In children it may be distinguished from chorea by the generally purposeful nature of the movements, their explosive, lightning-like character, their restriction to a group of muscles (the repetition of the same movement), the periods of freedom, and the associated psychic phenomena. Tic douloureux (page 808) and other reflex tics are of an altogether different nature. The outlook is not especially good, although recovery may ensue, especially in children. In the *treatment*, special attention should be paid to all possible sources of irritation and to the general health. Benefit attends proper educational procedures and exercises, and attention to the underlying neurotic condition.

CHOREIFORM DISORDERS.

Acute (Sydenham's) chorea, chronic hereditary (Huntington's) chorea, the habit spasms, the convulsive tics, and paramyoclonus multiplex are characterized by movements that more or less resemble one another and sometimes lead to diagnostic difficulties, but they should be readily differentiated, as already pointed out. In addition, there are the following choreiform affections: (1) *Senile chorea*, a disorder that occasionally develops in late middle life and much resembles Huntington's chorea, but differs in the absence of hereditary characteristics. (2) *Dubini's electrical chorea*, a rare disorder, characterized by rapid, violent, electric-like movements of the extremities, and sometimes also of the head and face; fever (perhaps infectious), ultimate atrophy of the muscles, and epileptiform convulsions are usually associated, and death supervenes after weeks or months. (3) *Henoch's hysterical chorea*, a rare disorder of adults, characterized by sudden rhythmic electric-like contractions of the muscles of the shoulder girdle, of the neck, of the abdomen (Salaam convulsion), or of the psoas muscles, etc. It is likely hysterical in nature, and may be associated with other hysterical stigmas. (4) *Saltatory spasm* (latah, myriachit, jumpers), a disorder characterized by the occurrence, when the subject attempts to stand, of strong contractions of the muscles of the legs and a consequent jumping or springing movement. It is most common in neurotic men, and may occur endemically: in Maine and Canada (jumpers), in Russia (myriachit), in Java and its neighborhood (latah).

FAMILY PERIODICAL PARALYSIS.

Family periodical paralysis is a rare disorder that occurs in families, is transmitted from one generation to another, involves both sexes, and is characterized by paralysis of all the muscles supplied by the spinal nerves. The nature of the disease is not understood; it is believed to be toxic, since attacks may be induced by fatigue, constipation, infections, etc.; but they often come on without definite cause. Mitchell, Edsall, and others have found a lessened creatinin excretion for several days before the attacks, and a normal excretion after the attacks. The symptoms develop rather insidiously, and consist of increasing lassitude and somnolence, and gradually progressing paralysis; this begins in the legs, and then involves the trunk and arms, and is usually complete at the end of twenty-four hours; the muscles innervated by the cranial nerves often escape entirely, but they may exhibit slight paresis. The paralysis is of the flaccid type; the tendon reflexes are lessened or lost; the superficial reflexes are diminished; and the electrical excitability of the nerves and muscles, especially to the faradic current, is diminished or lost. The heart may be dilated during the attack. Sensation and consciousness are

unimpaired. At the end of twenty-four hours or thereabouts, the paralysis begins to disappear, reversely compared to its development; recovery may be complete at the end of forty-eight hours. The attacks may recur at irregular intervals—days, weeks, etc., and they may disappear permanently in advanced adult life. The prognosis is good, although rarely death has occurred during an attack. Potassium citrate has sometimes shortened or aborted an attack; otherwise the treatment is symptomatic.

HEAT STROKE.

(*Sunstroke; Thermic Fever; Siriasis; Heat Exhaustion or Prostration; Insolation; Heat Cramps.*)

In consequence of exposure to excessive heat, one of three conditions may ensue—heat stroke, heat exhaustion, or heat cramps.

Heat stroke (*sunstroke, siriasis, thermic fever*) is a condition that occurs after exposure to the sun's rays, and develops especially in persons who at the same time are engaged in hard manual work, such as railway and other laborers, soldiers on the march, etc. There are no constant or characteristic lesions; congestion of the brain, parenchymatous degeneration of the solid viscera, undue fluidity of the blood, etc., have been found in fatal cases. The disorder is attributable to (toxic?) paralysis of the vasomotor centre in the medulla, provoked by the extreme or prolonged heat; some observers look upon it as an infection. The onset of the disorder is sudden. There may be premonitory headache, vertigo, nausea, vomiting, malaise, etc., but often the subject suddenly drops to the ground, becomes unconscious, goes into deep coma, and soon dies from vasomotor paralysis and heart failure; or he may be found in deep coma, with high fever (108° to 111° F., or more), hot skin, flushed face, rapid pulse, labored, stertorous, or Cheyne-Stokes breathing, and complete muscular relaxation; occasionally fibrillary twitchings of the muscles or convulsions occur. Subcutaneous petechiæ may be found, the pupils are usually contracted (although at first they may be, and sometimes they continue to be, dilated). Death may ensue within twenty-four hours; or the temperature may fall, coma disappear, and recovery ensue. Often, however, the patient thereafter is unable to withstand the effects of high temperature; and occasionally mental deterioration, with loss of memory, etc., may ensue.

Heat exhaustion, or prostration, is a condition that follows prolonged exposure to high temperatures (the sun's rays as well as indoor temperature), and it occurs especially in those doing hard manual labor, such as workers in iron foundries, stokers in steamships (among whom poor ventilation is doubtless a factor), soldiers on the march, etc. The condition is attributable to excessive sweating and insufficient water-drinking and consequent augmented specific gravity of the blood and inspissation of the tissues; a certain number of the cases, especially some, at least, of those occurring in the tropics, appear to be due in

part at least to long-continued exposure to excessive light (the actinic rays of the sun), whence they may be termed "light exhaustion or prostration." The symptoms consist of initial headache, restlessness and vertigo; and profuse sweating, subnormal temperature (96° F. or less), muscular cramps, prostration, and collapse. The surface of the body is cool, as well as bathed in perspiration; the pulse is small and rapid, and the breathing rapid and shallow; consciousness, aside from its obscuration by the general prostration, may be maintained, but delirium may supervene (whence the not uncommon suicidal tendencies and sudden jumping into the water of stokers of ocean steamships).

Heat cramps is a condition that develops in those who at the same time are exposed to great artificial heat and do much muscular work, especially workers in iron foundries, stokers on steamships, etc. It is described by Edsall as consisting of continuous fibrillary contractions of the muscles, especially those of the calves, and paroxysms of severe and painful, tonic spasms of the muscles, notably of the forearms and legs, but also of the abdomen, etc. The spasms occur spontaneously, and are provoked by efforts of voluntary movement and by mechanical, electrical, and other stimuli; and they last from one-half to one minute. An attack lasts, as a rule, not more than twenty-four hours, and is followed only by slight exhaustion and muscular soreness. Edsall attributes the condition to an acute degenerative process in the muscles.

Diagnosis.—The sudden onset of coma in those exposed to high temperature, and the high fever, serve, as a rule, to differentiate heat stroke from apoplexy and coma due to other causes; but one thus exposed, of course, may suffer an apoplexy or go into uremic or other coma, so that these must be excluded in all doubtful cases. Heat stroke, heat exhaustion, and heat cramps each is so distinctive that no difficulty should be experienced in differentiating them.

Prognosis.—The outlook in heat stroke is good if the patient is brought promptly under treatment; but in some cases death ensues almost immediately, or within twenty-four hours. The prognosis in heat exhaustion and heat cramps is good.

Treatment.—In heat stroke the bodily temperature should be reduced as promptly as possible—by cold-water or ice-water bathing, not forgetting to apply cold to the head; cold- or ice-water enemas also may be employed. Venesection is valuable in markedly asphyxiated subjects, and chloroform in the event of continued convulsions. Stimulants, such as strychnine, camphor, ammonia, whiskey, etc., should be employed in case of marked prostration. These, as well as external heat, are necessary in heat exhaustion. Enteroclysis and hypodermoclysis, with physiological saline solution, are valuable in all toxic states, including the cases of heat exhaustion and heat cramps, in which there has been much sweating and comparatively little water drinking. Morphine or apomorphine may be resorted to if necessary to control the painful myospasms.

VASOMOTOR AND TROPHIC DISORDERS.

ANGIONEUROTIC ŒDEMA.

(Quincke's Disease.)

Angioneurotic œdema is a disorder characterized by the occurrence of localized transient œdematous swellings of the skin and mucous membranes. It may be hereditary, and is believed by Quincke to be a vasomotor neurosis leading to increased permeability of the bloodvessels. Some attacks seem to be precipitated by articles poisonous to the individual, such as shellfish, certain berries, etc.; by ptomaines; by gouty, gastro-intestinal, or other autotoxic states, etc. The face, the limbs, the trunk, may be the seat of these swellings, which cause local redness, discomfort, pain, and itching; in the mouth and pharynx they may impede swallowing; in the larynx they may cause dyspnœa, stridor, and more or less asphyxiation; and in the gastro-intestinal tract they may give rise to more or less severe colicky pain, nausea, vomiting, and diarrhœa. They may be associated with erythema, urticaria, purpura, etc. (page 389). The attack may last only a few hours, or a day or two, and it may recur at regular or irregular intervals. The *treatment* is not very satisfactory. The intestinal tract should be thoroughly cleared by means of blue mass or calomel and saline cathartics; inquiry should be made into all possible exciting causes, especially articles of diet (which may be toxic to the individual and not to others); and nervines, tonics, such as strychnine, and so-called intestinal antiseptics, may be prescribed.

ERYTHROMELALGIA.

(Red Neuralgia; Weir Mitchell's Disease.)

Erythromelalgia is a disorder characterized by pain, tenderness, congestion, and increased temperature of the feet and sometimes of the hands, which develop especially when the parts are dependent. The disorder may apparently occur as an independent disorder—a so-called vasomotor neurosis; but similar phenomena may be associated with arteriosclerosis, multiple neuritis, etc. The symptoms begin with pain (usually burning in character) and tenderness in the feet, especially the soles; soon capillary distention supervenes and the feet become at first dusky red (often patchy, rather than uniformly); the arteries may pulsate visibly, the veins become dilated, and the local temperature become increased; but after some time the foot becomes purplish red, or of a pallid cyanotic hue, and cold. The hands are occasionally involved. The phenomena are aggravated by the dependent position, exercise, and warmth; they may be induced in susceptible subjects by long standing.

They may occur paroxysmally or become more or less permanent. A non-dependent position of the limbs, cool applications, massage, and electricity are of more or less avail, but the disease is usually quite intractable.

ACROPARESTHESIA.

Acroparesthesia is a painful disorder of the fingers and hands that occurs especially in women who do considerable washing (long immersion of the hands in hot water), and sometimes in men as well as women who use the hands a great deal. It is often associated with general ill health and impaired nutrition. It is perhaps sometimes of vasomotor origin, but it may also be neurotic, neuralgiform, or arteriosclerotic. The chief complaint is of numbness and tingling of the fingers and hand, which are likely to occur after prolonged immersion in hot water (therefore, toward night), or upon exposure to the heat of a fire, etc. Occasionally there is slight motor weakness and difficulty in executing fine movements (general involvement of the nerve). The symptoms vary in severity and duration. Usually they cease with cessation of the provoking cause, and they may be ameliorated by massage and electricity.

RAYNAUD'S DISEASE.

(*Local Syncope; Local Asphyxia; Symmetrical Gangrene.*)

Raynaud's disease is a disorder characterized variously by symmetrical syncope, asphyxia, or gangrene. In the early stage (local syncope) the fingers or toes, or both, and occasionally the ear tips or the nose, appear pale and cold—the so-called "dead" fingers and toes of the laity. This is analogous to the condition (chilblains) that may be produced by great cold (the low temperature of winter), and is attributed to spasm and incomplete obstruction of the peripheral arterioles. It may last for several hours or more and then subside, but in many cases a reaction ensues and the affected part becomes red or purplish, burning, tingling, and swollen (local asphyxia); sometimes exquisitely painful and more or less anesthetic. This is attributed to complete spasmodic obstruction of the arterioles and stagnation of the venous circulation; all fingers or toes may not become thus affected at once; one may remain pale and cold and its neighbor become cyanotic. In some cases the disorder consists of recurring attacks of such pallor and succeeding asphyxia of the fingers and toes; and the attacks may be set up by exposure to cold (worse, therefore, in winter), by emotional disturbances, by gastro-intestinal disorders (toxic?), etc. In other cases the lesions progress to local gangrene and sphacelation of the finger- and toe-tips; rarely, to more extensive loss of tissue, or multiple foci of gangrene in other parts of the body. In some cases there has been a curious association of these phenomena of Raynaud's disease and paroxysmal hemoglobinemia

(vasomotor disturbance in the kidneys?); these have occurred together, and in other cases the hemoglobinemia apparently has replaced the attack of the more familiar phenomena. The cause of the arterial spasm is not definitely known: Raynaud believed it to be of central origin; others attribute it to reflex or toxic causes; and others look upon it as syphilitic or as a phenomenon of arteriosclerosis and thrombo-angiitis. The *diagnosis* is readily made, and should exclude the cases due to manifest cardiac and arterial disease, ergotism, etc. The *treatment* should consist of a horizontal position of the limbs, wrapping the affected fingers and toes in cotton wool, and warm local applications. Nitroglycerin and other vasodilators, massage, and galvanism (the positive pole to the affected limb immersed in saline solution) are often of service. The pain may require the use of morphine or other narcotics and sedatives.

SECTION XI.

DISEASES OF THE MUSCLES.

MUSCULAR RHEUMATISM.

(*Myalgia.*)

Muscular rheumatism, or myalgia, is a disorder of the voluntary muscles and the related fasciæ, characterized by pain which is markedly aggravated by motion.

Etiology.—Muscular rheumatism is more common in men than in women; it occurs especially in those exposed to wet and damp, or directly to a draught of cold air (open window, etc.) and sudden cooling, particularly after exercise sufficient to induce sweating. In many respects it is analogous etiologically to, and may be the muscular equivalent of, rheumatic fever; at all events, it occurs in "rheumatic" and "gouty" subjects.

Pathology.—The nature of the disease is not definitely known. It is probably an inflammation of the fibrous investment of the muscular fibers and of the related fasciæ and periosteal attachments of the muscles; but some observers look upon it as a disorder of the sensory nerve endings in the muscles.

Symptoms.—The symptoms may come on suddenly or insidiously, and continue either a short or a long time (several hours to several weeks). The chief and often the only complaint is pain in and about a muscle or muscles; there is sometimes a little fever, but rarely any other general disturbance. The pain may be dull and aching, or sharp and cramp-like; it is markedly aggravated by any movement that involves the affected muscles, and it often, therefore, effectually prevents motion and leads to more or less contracture and malposition. Common types of the disorder are: (1) *Lumbago,* involvement of the lumbar muscles, which is usually very painful and often completely incapacitates the patient. One should exclude disease of the vertebræ, sacro-iliac joint, etc. (2) *Pleurodynia,* involvement of the intercostal muscles, and sometimes the pectorals or the serratus magnus. It is usually very painful, on account of the necessary respiratory movements of the chest. There may be circumscribed tenderness over the involved and contracted muscles. Pleuritis can be excluded by the absence of friction sounds;

and intercostal neuraliga, by the absence of pain following the course of the nerves, of acute unprovoked exacerbations of pain, and of the painful or tender spots (page 809). (3) *Torticollis* (wry-neck), involvement of the sternomastoid muscle and sometimes of other regional muscles (back of the neck especially). The sternomastoid muscle stands out as a tense, painful, and tender cord, and causes rotation of the head and chin toward the unaffected side.

Treatment.—In many cases prompt relief follows the use of remedies advised for rheumatic fever (page 256). In addition, rest to the disabled muscle or muscles is of the greatest importance. Pleurodynia is usually markedly benefited by strapping the side, or by a light application of the Paquelin cautery—which is also of the greatest service in lumbago. Hot applications, chloroform and other liniments, belladonna plasters, massage, galvanism, etc., afford relief in other cases. A hot or Turkish bath may cause almost immediate cessation of the pain. In obstinate chronic cases the iodides, mercury, belladonna, and iodine ointment, and attention to the dietary (antigout diet) are usually efficacious.

MYOSITIS.

Inflammation of the muscles is a rare disorder; but it may occur as a secondary, and apparently also as a primary, process. Secondary myositis may be: (1) Suppurative, in which case it is usually part of a general pyococcic process (page 61)), with multiple muscular foci, although rarely perhaps there may be only a single muscular focus. The involved muscle is at first swollen and painful, and gradually goes on to abscess formation; the abscess, if not evacuated, may lead to or increase the general pyococcic process. (2) Syphilitic. Early in the infection there may be a cellular myositis; late gummatous formation may occur. The gumma being absorbed gives rise to more or less loss of muscular substance and consecutive fibrosis. Primary myositis is very rare; occasionally it is observed as part of a dermatomyositis, a condition in which dermatitis and multiple involvement of the muscles and œdema occur. The muscles become tense, firm, and œdematous, and later degenerated and atrophic. In some cases hemorrhage may occur into the involved muscles (hemorrhagic polymyositis). The condition resembles trichinosis, but may be differentiated by the absence of eosinophilia and of the parasite in a piece of excised muscle.

PROGRESSIVE MUSCULAR DYSTROPHY.

(*The Muscular Dystrophies; Primary Myopathy; Pseudohypertrophic Muscular Paralysis.*)

Progressive muscular dystrophy is a primary disorder of the muscles characterized clinically by progressive muscular wasting.

Etiology.—Progressive muscular dystrophy is more common in males than in females. In most cases the disease is hereditary, being transmitted, as a rule, by the females to the males; in other cases it is familial, several members of the same generation being affected; some cases, however, exhibit neither hereditary nor familial characteristics. Infections, injuries, shocks, etc., have been variously looked upon as exciting causes, but at most these are of only secondary significance, the predisposition, the primary myopathy, being present.

Pathology.—The chief lesions consist of atrophy (which may be preceded by hypertrophy) of the muscular fibers, replacement fibrosis, and the deposition in the connective tissue of fat. The process may progress to complete atrophy of the muscular fibers, although the muscle spindles are likely to remain intact, and the deposition of fat may be so marked as to cause notable enlargement of the part (pseudohypertrophy; hypertrophic lipomatosis; pseudohypertrophic muscular paralysis). The nervous system usually exhibits no lesions; rarely, degenerated changes have been seen in the cells of the anterior cornua of the spinal cord, and functional or other changes beyond our present means of detection have been postulated. The disorder appears to be unquestionably a primary myopathy—a muscular abiotrophy; the individual is born with a muscular system ill adapted to the exigencies of life, so that sooner or later it begins to succumb and atrophy.

Erb has classified the cases into (1) those that begin in infancy and childhood, and (2) those that develop in adolescence or early adult life (between which there is no essential difference); and he has also classified them into (1) those in which there is a true hypertrophy of the muscular tissue, or a pseudohypertrophy (hypertrophic lipomatosis) of the part, and (2) those in which there is progressive atrophy of the muscle without the accumulation of fat; but between these cases there is also no essential difference, although the distinction is convenient clinically.

Symptoms.—The disease usually begins before puberty, but may be delayed until adolescence or early adult life (before the twenty-fifth year). The onset is insidious, the first symptoms being muscular weakness and disability, with or without increase in the volume of the muscles of the part. Depending upon the muscles involved, several clinical types of the disease are distinguished, although there is no sharp line of demarcation between them: (1) *The pseudohypertrophic form of Duchenne.* This usually develops before the tenth year and in males. It involves especially the muscles of the calves—which become much enlarged. The child in walking readily grows tired, stumbles, or drags the legs (in marked contrast to the healthy looking calves); and, to aid progression, soon begins to bring the pelvis into play, and develops a waddling gait. The muscles of the thigh, buttocks, and back soon become involved; the child experiences increasing difficulty in arising from a lying to a standing posture, and characteristically aids himself by "climbing up on his legs with his hands." When erect, he stands with his legs apart, his shoulders thrown back (in an effort to compensate the forward curvature of the

spine, due to atrophy of the lumbar muscles), and the abdomen protruded. The common enlargement of the muscles of the calves and of the anterior aspect of the thighs and the atrophy of the glutei muscles accentuate this attitude. Later, the muscles of the shoulder girdle may become involved. Muscles originally hypertrophied become atrophic, and contractures may ensue. Cases unassociated with the pseudohypertrophy are sometimes spoken of as the Leyden-Möbius form. (2) *The scapulo-humeral form of Erb (Erb's juvenile form).* This begins, as a rule, between the ages of twelve and sixteen years, and involves first the muscles of the shoulder girdle, and then those of the upper arm and the anterior chest, and finally those of the back. In advanced cases the muscles of the thigh and calf become involved, so that the condition much resembles the pseudohypertrophic form of the disease. (3) *The facio-scapulo-humeral type of Landouzy and Dejerine.* This begins in early childhood, in the third or fourth year, as a rule, and is especially characterized by atrophy of the muscles of the face, as well as of the muscles of the shoulder girdle and the upper arm (Erb's infantile form); the pseudohypertrophic type of Duchenne also may be associated. The lips protrude and cannot be closed, and their finer movements (articulation, puckering, retaining fluid and food) are interfered with, although mastication and deglutition may be unimpaired. The ocular and palpebral muscles often escape.

Diagnosis.—The different forms of muscular dystrophy are readily distinguished by their hereditary or familial occurrence, their usual onset in early life, involvement of the muscles near to the trunk (rather than the small muscles of the hands and feet, as in progressive muscular atrophy), and the occurrence of pseudohypertrophy of certain muscles, especially the calves; by gradual reduction in the deep reflexes and electrical excitability of the muscles—depending upon the gradual muscular atrophy (the muscular fibers remaining continuing to respond); and by the absence of the reactions of degeneration, of fibrillary twitchings, and of sensory changes. These serve to exclude chronic poliomyelitis, progressive muscular atrophy, and multiple neuritis.

Prognosis.—Muscular dystrophy is slowly progressive, but it may last twenty or thirty years, and does not lead directly to death.

Treatment.—It is at least doubtful whether any treatment is really efficacious. Regulated exercises, passive movements, massage, electricity, hydrotherapeutic measures, and tonics are serviceable in some cases.

MYOCLONUS.

(*Paramyoclonus Multiplex, Friedreich.*)

Myoclonus is a disorder characterized by clonic, constant or paroxysmal, contractions of the muscles, chiefly of the legs. The disease is sometimes hereditary; it may affect several members of the same family; it occurs especially in males, and it may be found in epileptic subjects.

Its cause is not known; it is said to follow fright, shock, and general ill health. Its nature has not been determined; some observers believe it to be allied to the convulsive tics, electric chorea, etc. The characteristic *symptoms* consist of sudden electric-like, clonic contractions of the muscles, especially of the legs, sometimes of the trunk, and rarely of the face. The spasms, as a rule, are bilateral and rhythmic; they vary from 50 to 150 per minute, and may be so violent as to cause motion of the entire limb, or even tossing about of the patient. They are often lessened by voluntary movement, and they usually cease during sleep. In the paroxysmal cases tremors of the muscles may occur during the intervals. The reflexes are usually exaggerated, the nutrition of the muscles is well maintained, and there are no reactions of degenerations, sensory changes, or psychic disturbances. The disorder may be distinguished from acute chorea by the sudden electric-like quickness of the movements and their occurrence especially in the legs, and from the convulsive tics, the movements of which are purposeful and usually unilateral. The disorder lasts a variable time, and may cease spontaneously. The *treatment* consists of the use of sedatives, such as the bromides, valerian, hyoscine, etc.

CONGENITAL MYOTONIA.

(*Thomsen's Disease.*)

Congenital myotonia is a disease characterized by rigidity or tonic contractions of the muscles upon attempting voluntary movements after a period of rest. It is usually hereditary; it may affect several members of the same generation; it is almost ten times as common in males as in females, and it usually develops during childhood. The nature of the disorder is not known; it has been attributed to changes in the muscle fibers, to an auto-intoxication involving especially the muscles, etc. The nervous system shows no lesions. The *symptoms* consist of the occurrence of tonic spasms of the muscles upon attempting voluntary movements—such as arising from a chair, walking, grasping an object, closing the eyelids, etc.; that is, the patient experiences difficulty and delay in initiating the movement, but, once begun, persistence in the movement results in relaxation of the contracted muscle; but a period of rest is followed by recurrence of the phenomena. This incapacitates the child more or less and prevents his engaging in children's plays and games. The muscles are usually well developed and may even appear hypertrophied, but some muscular weakness is common, although there is no atrophy. The mechanical excitability of the muscles is increased—a blow, for instance, causing a quick and lasting contraction (ten to twenty seconds). The electrical excitability also is increased and occasions the so-called myotonic reaction: undue excitability to mild faradic and galvanic currents; rather slow maximum response to the current and slow relaxation; under the influence of the galvanic current the anode-closing contraction (ACC) becomes equal to or greater than the cathode-closing

contraction (CCC); and if a continuous galvanic current is passed through the muscle, rhythmic, vermicular contractions pass from the cathode to the anode. No efficacious *treatment* is known.

CONGENITAL AMYOTONIA.

(*Congenital Myatonia; General or Localized Hypotonia of the Muscles of Children; Congential Hypotonic or Atonic Pseudoparalysis; Oppenheim's Disease.*)

Congenital amyotonia is a disease characterized by diminished tone (hypotonia) of the voluntary muscles. The disorder is undoubtedly congenital, and becomes manifest during childhood; infections are sometimes said to act as exciting causes. The nature of the condition is not known; changes in the muscles analogous to those of the muscular dystrophies have been found. The *symptoms* consist of flaccidity and loss of tone and power of the voluntary muscles, but there is no absolute paralysis and no wasting of the muscles. The condition is usually most marked in the limbs, and is often associated with relaxation of the joints, so that the limbs may be readily placed in grotesque positions and luxations may ensue; but the muscles of the trunk and of the face may be involved. The deep reflexes are lessened or lost; the electrical excitability is diminished, and currents much stronger than in health can be borne with equanimity. The disorder may be distinguished from the muscular dystrophies by its being congenital (but not hereditary) and non-familial, and by the marked hypotonia and absence of wasting of the muscles. It should be readily differentiated from acute poliomyelitis, rickets, etc. The prognosis is good, since the condition tends to lessen and finally to disappear as the child grows older, but contractures may occasionally occur. The *treatment* consists of hygienic and dietetic regulations designed to improve the general health and nutrition of the child, and of massage, electricity, and hydropathic measures to increase the tone of the muscles.

SECTION XII.

DISEASES OF THE BONES AND JOINTS.

ACUTE ARTHRITIS.

Etiology.—Acute arthritis, aside from, and often including, the cases that follow local injury, is always due to bacterial infection or intoxication. It may occur as a primary disorder, of which the joint manifestations are the conspicuous feature—rheumatic fever (page 253); or it may occur in the course of or following any of the well-known pyococcic, pneumococcic, gonococcic (page 91), influenzal, and other infections. Special importance attaches to unsuspected, masked, and more or less obsolete infections, such as tonsillitis, pyorrhœa alveolaris, chronic gonococcic infection with involvement of the seminal vesicles and the prostate, other infections of the urinary tract, of the gastro-intestinal tract, etc.

Pathology.—One or several joints may be involved. In the mild cases the lesions consist of congestion, swelling, and œdema of synovial tissues, and the outpouring into the cavity of the joint of a more or less turbid albuminous fluid containing a few flakes of fibrin and leukocytes. In more severe cases the synovial and fibrous capsules of the joints are swollen, œdematous, and adherent, and the cavity of the joint is filled up, perhaps obliterated, by more or less thick, fibrinous, fibrinopurulent, or distinctly purulent exudate; the articulating cartilaginous surfaces may reveal erosions with thickened infiltrated borders. In less severe and more chronic cases there may be more or less villous proliferation of the synovial tissue. The infecting microörganisms may sometimes be isolated from the articular fluid or tissues, but often having initiated the lesions they die out; or the lesions may be set up by bacterial toxins alone.

Symptoms.—The onset of the disorder may be abrupt or more or less insidious, depending upon the severity of the infection. The chief symptoms consist of pain, tenderness, swelling, increased temperature, and disability of the joint or joints. On palpation the joint is soft and boggy—in consequence of more or less intra-articular fluid, swelling of the synovial tissues, and periarticular œdema. In addition, there are the general evidences of infection—due in part to the primary disorder, but perhaps aggravated by the local joint condition—fever, rapid pulse, and leukocytosis; in the event of suppuration, also chills or chilliness and sweats.

Diagnosis.—The diagnosis is quite apparent from the local conditions. Efforts should be made to determine the nature of the infection—by searching for sources of infection and cultivating therefrom the infecting microörganism, or, when the conditions appear to warrant it, aspirating the joint and studying the fluid bacteriologically and cytologically.

Prognosis.—The prognosis depends upon the severity of the infection and the nature of the ensuing lesions. There is always a tendency for the condition to become polyarthritic, even in originally monarthritic cases, and in all but the mildest cases more or less permanent disability is likely to ensue.

Treatment.—All possible local sources of infection anywhere in the body must be eradicated, since the joint symptoms may be kept active by bacterial toxemia without local joint infection. The best treatment comprises rest and fixation of the joint and the local remedies mentioned on page 257. The rest should not be too long continued, passive movements being substituted as soon as possible. Good results also may attend the use of specific bacterins. In cases of severe and progressive infections, resort should be had to surgical procedures—opening and irrigating and draining the joint.

CHRONIC ATROPHIC ARTHRITIS.

(*Arthritis Deformans; Rheumatoid Arthritis; Chronic Polyarthritis of the Atrophic Type of Goldthwait.*)

Etiology.—The form of chronic polyarthritis known as deforming or rheumatoid arthritis (the atrophic type of Goldthwait), is a disease of adult life, and is more common in women than in men. Goldthwait discounts the likelihood of an infectious cause in these cases, emphasizing the etiological significance of poverty, wear and tear of life, grief, fear, severe nervous shock, and great physical and mental strain. These are undoubtedly present in many cases, and of unquestionable causative importance; but in many cases also, and in increasing numbers as the cases are the more carefully studied, some chronic unsuspected, masked, or concealed infection is found—in the tonsils, about the teeth, gleet, chronic gastro-intestinal infections, etc.; some cases represent an advanced stage of a disorder developed acutely and undoubtedly infectious; and Still has described a type of the disorder occurring in children, and characterized by enlargement of the lymph nodes and the spleen, in addition to the joint lesions. In some cases gastro-intestinal and other metabolic poisons may be an active factor.

Pathology.—The lesions involve all the articular structures—the synovial membrane and fluid, the cartilages, and the bone. The earliest changes consist of inflammatory and hypertrophic alteration of the synovial tissue, with the production of a condition called villous arthritis, and thickening, swelling, and infiltration of the peri-articular tissues. Gradually the inflammatory lesions of the synovial tissue subside, and cicatri-

zation and atrophy ensue. In the course of time the cartilage becomes opaque, striated, and atrophied, so that large or small erosions of the articular surfaces develop; opposed surfaces of exposed bone being brought into contact, give rise to rough, grating impressions; the bone trabeculæ become reduced in size and number, and other atrophic and rarefying changes ensue. The synovial fluid may be present in excess in the early stages, but late it is usually normal in amount, the increase in the size of the joint being due to thickening of the soft periarticular tissues. Osseous spurs may sometimes be seen (attempt at repair).

Symptoms.—Chronic atrophic arthritis is a polyarthritic disorder that begins insidiously in the small joints of the hands—not always at the same time in both hands, but both soon become involved; later, the small joints of the feet, and the wrists, elbows, ankles, knees, shoulders, and other joints, even all the joints of the body, may become affected. A single joint is rarely involved alone. The patient at first experiences pain or difficulty in executing fine and complicated movements involving the hands; the phalangeal and metacarpophalangeal joints soon become swollen—due to periarticular œdema and infiltration, in part sometimes to increase of the synovial fluid. Deformities gradually ensue, which result in part from the swelling, in part from gradual atrophy of the articular tissues, and in part from subluxation and flexion (the stronger flexors overcoming the weaker extensors); lateral deformities also may ensue, notably ulnar deviation of the hands and knock-knees. Crepitation occurs on flexing and extending the joints (due to the rubbing of villi or erosions), and more or less fixation may result from the contraction of muscles or bony ankylosis. The muscles about the joints usually atrophy. Fluctuation in the severity of the lesions occurs from time to time; and the condition may even abate or become quiescent. There are, as a rule, few general symptoms aside from general malnutrition and anemia.

Diagnosis.—The symptoms are sufficiently distinctive to enable one to recognize the disorder. The lesions may be well studied by radiographic examination.

Prognosis.—The outlook is not promising, although considerable amelioration of the condition may be brought about in the early stages.

Treatment.—Sources of infection must be searched for and eradicated; and attention must be given to improving the general nutrition of the patient by good food, good air, rest, and freedom from distress and worry. Internally, syrup of iron iodide, cod-liver oil, arsenic, and thyroid extract are sometimes efficacious. Locally, belladonna, mercury, and iodine ointment, massage, electricity, passive movements, hydrotherapeutic measures, exposure to superheated air ("baking" the joints) render service. If there is much and persistent accumulation of fluid, it may be withdrawn. Care must be taken that flexions, subluxations, and other deformities do not ensue, and if ankylosis seems inevitable, that it occurs while the joints are in a position to permit of their being useful. Persistent and disabling deformities may be corrected by surgical methods.

CHRONIC HYPERTROPHIC OSTEO-ARTHRITIS.

(*Arthritis Deformans of the Hypertrophic Type of Goldthwait.*)

Etiology.—Goldthwait applies the term chronic hypertrophic arthritis to a "chronic constitutional disease with local joint manifestations, which may or may not be polyarticular." The disorder is more common in men than in women, and occurs in middle life and old age. The etiological factors are not well understood; some significance is attached to exposure; to injuries, which may be single and severe and, involving one of the large joints, may apparently soon lead to the disease, or which may be slight, inconsequential, but frequently repeated and related to the patient's occupation; to gastro-intestinal derangements and consequent auto-intoxication; and to the changes of senility.

Pathology.—The joints most commonly involved are the fingers, knees, vertebræ, hips, and feet. The chief lesions consist of slight localized villous hypertrophy of the synovial tissue and of the related cartilage; and of hypertrophy of the cartilage along the outer margin of the joint and that associated with the ligaments (so-called lipping). This hypertrophy consists of fairly normal cartilage (in contrast with the changes in the cartilage in the atrophic form of the disease); it is quite irregular in degree, and soon becomes ossified—that is, into it osteophytes develop (so-called Haygarth's nodosities at the knuckles); sometimes there is an associated bony spur formation or hyperplasia at the point of attachment of the ligaments and tendons. These together produce more or less joint disability and fixation. Erosions occur, but they result from the action of the osteophytes or from pressure, and they differ from those of the atrophic type of the disease in showing at their bottom eburnated cartilage and not exposed bone.

Symptoms.—The onset of the disorder is slow, and considerable progress may be made before noteworthy symptoms supervene. The usual complaint is of articular discomfort and pain and more or less disability, all of which are increased on activity and improved on resting. From time to time the symptoms disappear, perhaps entirely, and leave no symptoms except the bony deformities. The condition is quite common in the hands, occurring as small, hard knobs of hypertrophied cartilage and bone at the dorsal or dorsolateral margin of the articulating surfaces of the terminal phalanges (*Heberden's nodes*). Flexion or lateral deviation of the terminal phalanges occurs from pressure atrophy on the side toward which the nodes push. The condition is incurable, but it brings with it the happy reflection that its subjects rarely become affected with disease of the larger joints. In the knees, considerable enlargement, of the femur, the tibia, and the patella, with subluxation and fixation of the joint (osteophyte formation) ensue, and the patient is often permanently disabled. In the hip (morbus coxæ senilis) the changes involve the head of the femur and the acetabulum; the acetabulum becomes broadened and the upper lip grows over and may completely surround the neck of

the femur, effectually limiting motion. In the vertebræ the lesions often involve several adjacent vertebræ; the cartilaginous proliferation may extend to several neighboring bones, and the osteophytic proliferation may invade not only this but also the intervertebral cartilages, and may extend down the anterior and anterolateral aspects of the vertebræ, effectually ankylosing them (so-called spondylose rhizomelique). The condition is most common in the thoracicolumbar and the cervical vertebræ. It gives rise to more or less, often marked, rigidity of the vertebræ, and local pain; the pain often radiates (unusually unilaterally) to the area of distribution of the sensory roots that enter the vertebral column amidst the lesions.

Diagnosis.—The disease is readily recognized. The articular lesions can be well studied by radiographic examination.

Prognosis.—The outlook as to cure is not good. Once established the disease is irremediable, but some amelioration can be effected in the early stages.

Treatment.—The treatment is that recommended for the atrophic form of the disease. Care should be taken to prevent deformities; if they develop they may be corrected by surgical methods.

OSTEOMALACIA.

(*Mollities Ossium; Fragilitas Ossium.*)

Osteomalacia is a chronic disorder of nutrition characterized by decalcification and progressive softening of developed and ossified bones, leading to bending or fracture and more or less deformity.

Etiology.—The disorder is rare, but occurs, especially in adult life and in women (10 to 1 man). It is commoner among the poor than the well-to-do, and seems to be epidemic in certain regions (in Europe). It occurs most frequently in pregnant and nursing women, especially multipara, but also in virgins (relation to ovulation?); pregnancy may merely cause an exacerbation of the disease already existent.

Pathology.—The bones most commonly affected are the pelvic bones, the vertebræ, and the ribs; the long bones usually are affected late. The lesions consist in decalcification of the compact and spongy parts of the bone and their replacement by a soft, vascular, gelatinous, sometimes hemorrhagic or cystic, tissue; the periosteum may be involved or thickened and hyperemic. The bones in consequence become softened or brittle, and, yielding to divers influences, become bent, distorted, and may be readily fractured. The nature of the disease is not known; a relationship between the osteomalacia (decalcification) of pregnancy and lactation and the need of the growing fœtus and child for lime and phosphorus has been postulated. An influence of the ductless glands also has been suggested; the parathyroids are known to have an influence on calcium metabolism (deficiency provoking tetany); the ovaries perhaps influence the absorption of calcium (and some cases of osteomalacia

have been cured by oöphorectomy); the thyroids also have been implicated—but the exact interrelationship, if any, is not well understood.

Symptoms.—The onset of the disease is insidious, and it may be far advanced when first recognized (as, for instance, vaginal examination at impending parturition). The earliest symptom usually is pain (misinterpreted as rheumatic) in the pelvis, vertebræ, thorax, legs, etc., perhaps associated with difficulty in locomotion (hobbling or wobbling gait). Gradually deformities develop; the sacral promontory and the acetabula approach one another (osteomalacic pelvis); lordosis, kyphosis, or skoliosis ensues; the bones of the legs become curved (the patient often is obviously growing shorter), and fractures may occur spontaneously or on very slight provocation. The cranium usually is not much, if at all, involved. The patient may become bedridden. The muscles are usually flaccid and atrophic (rarely spastic), and may exhibit fibrillary twitchings, intention tremors, etc. The urine contains an excess of calcium and phosphorus.

Diagnosis.—The diagnosis often is not made until the disease is well advanced—when it presents no difficulties. In the early stage, muscular rheumatism and divers diseases of the bones and of the nervous system must be excluded. Attention to the possibility of the disease in a pregnant or nursing woman, examination especially of the pelvic bones for deformities, radiographic examination (for decalcification), and a study of the calcium output in the urine should enable the diagnosis to be made early in many cases.

Prognosis.—The disease is usually progressive, running a course of five or ten years or more; sometimes the course is rapid—less than a year; sometimes it becomes arrested, and perhaps made worse by a subsequent pregnancy. Death usually ensues from progressive weakness or intercurrent infection.

Treatment.—The treatment is largely symptomatic: good food, good hygiene, and the avoidance of etiological factors—nursing in those already affected, as well as subsequent pregnancy. Calcium and phosphorus (cod-liver oil and other phosphorus-bearing oils and foods) often are markedly beneficial. Improvement has followed the use of adrenalin to the point of toleration. Ovariotomy has led to improvement in advanced cases in women.

OSTEITIS DEFORMANS.

(*Paget's Disease.*)

Osteitis deformans is a rare disorder, characterized by "enlargement and forward projection of the head, dorsocervical kyphosis, prominence of the clavicles, spreading of the base of the thorax, a diamond-shaped abdomen crossed by a deep sulcus, a relative increase in the width of the hips, and an outward and forward bowing of the legs" (Packard, Steele, and Kirkbride). The subjects become notably reduced in height.

The cause of the disease is not known; it is believed to be related to osteomalacia, and perhaps to disorders of the pituitary body and the other ductless glands. The lesions consist of a rarefying and formative osteitis. The diagnosis is apparent at a glance. Treatment seems to be inefficacious.

HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY.

Hypertrophic pulmonary osteo-arthropathy is a rare disorder, characterized by enlargement of the ends of the bones of the hands and feet, especially the terminal phalanges, and sometimes also of the distal ends of the long bones of the forearm and legs. The ends of the fingers become characteristically bulbous and the finger nails elongated and curved about them. The nature of the disease is not known. Most of the cases are associated with long-standing (usually suppurative) disorders of the lungs or pleura, and it has been supposed (Marie) that the toxins of the pulmonary disorder, reaching the bones by way of the circulation, set up an ossifying periostitis. A somewhat similar condition sometimes occurs in chronic heart disease, syphilis, tuberculosis; or it may develop from unknown causes. Freedom from involvement of the bones of the skull and face readily excludes akromegaly.

OSTEOGENESIS IMPERFECTA.

Osteogenesis imperfecta is a rare disorder, characterized by imperfect development of the bones, especially of the skull and the extremities. The bones are exceedingly fragile, so that intra-uterine and extra-uterine fractures are common. Should the child survive, ossification ultimately may become normal, although the bones may be more or less deformed.

ACHONDROPLASIA.

(*Chondrodystrophia Fœtalis.*)

Achondroplasia is a disorder due to perverted development (dystrophy) of the epiphyseal cartilages (connective-tissue invasion from the periosteum), in consequence of which the epiphyses and diaphyses become prematurely united and the normal growth of the long bones does not occur. The subjects are dwarfs, usually with normal heads and trunks, and short, stumpy extremities.

FACIAL HEMIATROPHY.

Facial hemiatrophy is a rare disorder, characterized by atrophy of the bones and soft tissues of one-half of the face. The nature of the disorder

is not well understood; interstitial neuritis of the entire trigeminal nerve has been found. The disorder begins usually in childhood, although it may come on later; and it may follow an injury or infection. There is gradual wasting of all the tissues, skin, subcutaneous connective tissues, and the bone, and to some extent also the muscles. The skin becomes pale, the hair whitens and may fall, and the teeth may drop out. There may be some associated sensory changes. The condition is readily recognized.

LEONTIASIS OSSEA.

Leontiasis ossea is a rare disorder, characterized by enlargement of the cranial, and sometimes the facial, bones—occasioning a so-called leontine expression. The nature of the disease is unknown.

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